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and
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for their pioneer efforts and
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Program on pages 249-251

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Dr. Clarke Chamberlain is well known to all periodontists because of his activities in the Academy and as Vice Chairman of the American Board of Periodontology. His activities are too numerous to mention; suffice it to say, he has been on numerous committees and held many offices in dental organizations. In addition he is active in community affairs and belongs to the Royal Order of Jesters, Masonic Lodge, and Rotary Club. He is married and has three fine children. The American Academy of Periodontology is deeply indebted to him for his efficient work as secretary.

THE JOURNAL OF PERIODONTOLOGY

OCTOBER, NINETEEN HUNDRED FIFTY-ONE

Experimental Evidence of Systemic Influence on the Development of Periodontal Disease

By PAUL E. BOYLE, D.M.D.*†

VOLTAIRE is credited with saying "If you would argue with me, define your terms." Disease is a disturbance in function or structure—a lack of ease or of the comfortably working together of body structures. Inherent in the concept of disease is the tendency of the cells, tissues and organs to return to the harmoniously functioning state called health—part of the *Wisdom of the Body* to use Cannon's[1] phrase. Periodontal disease is either an inflammatory or a degenerative process, or a combination of both, involving the supporting structures of the teeth.

Systemic influences are contrasted with local influences—factors such as nutritional and hormonal effects which affect local tissue cells by way of the circulation. Experimental evidence consists of the results of a series of carefully conducted observations under planned conditions. This does not necessarily involve the use of laboratory animals though much of the work discussed in this paper is of that nature. Carefully controlled clinical observations may be a most valuable kind of experimental evidence. The picture of clinically healthy gums and periodontal tissues is well known. The gingival crevice is of minimal depth, the marginal gingiva knife edged and the attached gingiva firm, pink and stippled. In such a mouth an overhanging filling or poor contact point leading to food impaction causes an inflammatory reaction. The irritation gives rise to swelling, a change in color, proliferation of epithelium with thinning and a tendency to bleed on slight pressure. Once the local condition is corrected the tissues quickly return to normal. If, however, the condition is not controlled there may be loss of the alveolar crest and eventually local pocket formation. The inflammatory process is accompanied by resorption of bone. This is an invariable sequence whether in periapical infection, osteomyelitis or periodontal irritation. The responsibility of the dental profession for preventing local periodontal disease, both by competent, conscientious, restorative work and by removing local causes of irritation, is a very real responsibility.

Occasionally a patient is observed who for some time has avoided chewing on one side of the mouth, perhaps because of a sensitive tooth. The gums on the side in use may be firm, pink and healthy. On the unused side the gums appear flabby, red and swollen and they bleed at a touch. When examined carefully,

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†Read before the Second Annual Berkshire Conference, June 13, 1951.

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calculus is found under the gingival margins. On roentgenographic examination, the alveolar bone is less dense and the alveolar crest may be irregularly resorbed. Observations of this kind have suggested the value of vigorous mastication and of simulated exercise by proper use of the toothbrush and other devices. Experiments with laboratory animals have indicated the value of vigorous mastication in maintaining the health of the periodontal tissues.[2]

On the other hand, excessive forces applied to one or more teeth may also be associated with the development of periodontal disease. In experimental animals, crushing forces cause necrosis of the periodontal membrane followed by resorption of the alveolar bone and movement of the tooth to a more favorable position.[3] This normal ability of local tissues to compensate for excessive stress suggests that systemic factors to be considered later are of particular significance in periodontal disease which develops in regions subjected to traumatic occlusion.

Next consider the patient whose gums are uniformly inflamed. Metallic poisoning (lead, mercury, bismuth) may give such a picture. In the days when mercury and bismuth were used in the treatment of syphilis it was recognized that expert periodontal care by the dentist was of great importance in enabling the internist to continue his treatment. The interrelations between local and systemic factors are obvious here.

Let us look more closely at the periodontium. The tissues which surround the tooth consist of epithelium, connective tissue and bone together with their vascular and nerve supply. The epithelium normally is firmly adherent to the tooth surface and is tightly bound to the underlying alveolar bone by connective tissue fibers. Fibers also run from tooth to tooth as the transeptal fibers and within the alveolus they constitute the principal fibers of the periodontal membrane. The attachment of tooth to bone is slightly movable and the articulation is technically termed a gomphosis.

Epithelial tissues cover and protect other tissues. Infolded they also form the glands both of external and internal secretion. The gingival epithelium is specialized to withstand friction. Its lowermost cells proliferate in a direction polarized toward the outer surface. The deeper ones remain behind as perpetual mother cells, dividing again and again while the daughter cells move toward the surface and are eventually cast off. Cells of the intermediate layers are bound together by prominent fibrils and cementing substance and are commonly called prickle cells. As they approach the surface the fibrils are lost and refractile granules appear. Finally the nucleus shrivels up and disappears and the surface cells become hornified scales. The pale pink color of firm healthy gums is due to the semitransparent nature of the surface scale cells together with the dispersion of light by the granules in the next deeper layer of cells so that the red color of the underlying blood vessels is dispersed and modified. The vestibular (alveolar) mucosa lacks the hornified and granular layer of cells. A deep red color is normal in this region although in the gingiva proper it indicates pathologic change.

The phenomenon of cells dying in order to function fully is an extraordinary one recognized and described by the late Charles Sedgwick Minot[4] under the name cytomorphosis. Remember that human red blood cells also must lose their nuclei and die in order properly to transport oxygen.

Cancer, leukoplakia and lichen planus are pathologic alterations of the normal cyclic changes of epithelial cells. The simplest view of the common squamous cell cancer of the mouth is that the dividing mother cells grow inward invasively instead of remaining at a constant distance from the surface. Leukoplakia and lichen planus, on the contrary, are characterized by an abnormal piling up of scale cells upon the surface. The etiology of these conditions is unknown although they are

obviously variations in the normal growth and differentiation pattern of epithelial cells.

Vitamin A has been shown to be essential for the normal function of epithelial cells. The story is most complete for the rods of the eye which allow vision in semidarkness. Vitamin A is part of a cell enzyme system by which visual purple is regenerated. In specialized epithelia such as those of the tooth germ, salivary gland and respiratory tract, the mother cells continue to proliferate in vitamin A deficiency. Their daughter cells, however, as they move toward the surface, fail to differentiate in normal fashion but form prickle and scale cells. When vitamin A is restored to the diet the mother cells continue to proliferate but now the new daughter cells as they approach the surface again differentiate normally into ameloblasts, gland cells, etc.[5]

The change in the gingival cells in vitamin A deficiency is less obvious. There is, however, a tendency for the surface scale cells to accumulate in great masses—a sort of dandruff of the gums. This cell debris acts as a culture medium for bacteria and in laboratory animals fairly typical pyorrhea pockets with eventual loss of the teeth has been shown to occur in vitamin A deficiency.[6] As Glickman[7] has shown in vitamin C deficiency, there is always a local factor initiating the development of the periodontal disease—the deficiency state sets the stage for the local process to occur earlier and to progress more rapidly than it would otherwise. Foreign particles such as oat husks and hair are the usual initiating factors.

Certain hormones also have a marked effect on epithelial tissues. This is most striking in the case of the estrogens produced by the mature ovary. The vaginal mucosa undergoes cyclic changes which, as Wolbach and Howe have emphasized, closely resemble the changes of vitamin A deficiency and repair. During estrus the surface cells become cornified and adapted to withstand friction. They are also more resistant to bacterial infection. In diestrus the scale cells are cast off and a more delicate surface layer of living cells remains.

Similar though less striking changes occur in the oral mucosa. The tendency of the gums to bleed in females during menstruation and pregnancy has long been recognized. Ziskin[8] studied these changes in normal and castrated monkeys. With lack of estrogens the gingivae were poorly hornified and when these hormones were injected, the scale cells reappeared and the gums again became pink and firm. Desquamative gingivitis usually occurs in females after the menopause and some success in treatment with estrogens has been reported. Likewise a degree of favorable response has been demonstrated in the treatment of leukoplakia with estrogens.[9]

Thus it appears clear that both vitamin A and the female sex hormones play a role in the normal physiology of the epithelial cells of the gingivae and therefore in the maintenance or restoration of healthy periodontal tissues. What the interrelations of these substances are or just how they influence cell physiology and chemistry or the mechanisms by which invasion by infectious organisms is promoted or retarded largely remain as problems for future research.

The connective tissues which underly the epithelium and which form the periosteum and the periodontal membrane are composed of cells, but cells of a very different morphology and physiology These are stellate cells with long, motile protoplasmic processes. In tissue culture they can be watched extending and retracting these processes as an ameba does. Unlike epithelial cells, connective tissue cells are widely separated by intercellular substance. This substance under the microscope is of a glassy or hyaline nature. Recently it has been shown to consist of a complex carbohydrate to which the name hyaluronic acid has been given. Imbedded in the matrix are fibers of several types of which collagen fibers are the most numerous. They

consist of chrondroitin-sulphuric acid-protein combinations. These fibers are the ones which form the principal fibers of the periodontal membrane.

Enzymes have been discovered which will depolymerize (liquefy) the ground substance (hyaluronidase) and the collagen fibers (collangenase). Hyaluronidase has been shown to be formed by infectious organisms and to be correlated to a degree with the invasiveness of these organisms. It is obvious that the bacterial flora of the pyorrhea pocket should be investigated with reference to the role of hyaluronidase in connective tissue invasion and liquefaction.[10]

Epithelial tissues of malignant tumors have been alleged to possess collagenases and their invasive powers can logically be attributed to these enzymes. Many students of periodontal disease have felt that the proliferation of the epithelium to line a periodontal pocket is an active, invasive process by which the connective tissue fibers are liquefied and destroyed. Others have considered the periodontal pocket as being similar to ulcers elsewhere. Active infection leads to death of the connective tissue cells and the enzymes produced by leukocytes cause dissolution of the collagen fibers. The surrounding epithelium, as always, is stimulated to grow in an attempt to repair and recover the ulcer surface. In the periodontal pocket a subacute or chronic infection becomes established which is progressive, leading to eventual loss of the tooth unless the pocket is obliterated. Whether or not the epithelium of the periodontal pocket possesses collagenases is a problem on which we must await definite information from further experimental observations.

The fact that vitamin C is essential for the formation of collagen fibers is one of the great contributions of Wolbach and Howe.[11] In vitamin C deficiency (scurvy) connective tissue cells proliferate but are unable to produce normal intercellular substances. Up to this point we have been considering connective tissues of the soft variety. Bone, dentin and cementum are closely similar connective tissue matrices which have become infiltrated with calcium salts. In vitamin C deficiency, bone, dentin, and cementum fail to form properly, although the process of calcification which we shall discuss subsequently is not disturbed. Loosening of the teeth has been recognized for centuries as a cardinal sign of scurvy. In experimental animals, long continued administration of diets containing minimal amounts of ascorbic acid but otherwise adequate have been shown to result in an inability of the periodontal tissues to withstand functional stress[12] which as you remember is the cardinal sign of the systemic type of periodontal disease as described by Gottlieb.[13]

Vitamin D aids in the absorption of calcium and phosphorus from the intestinal tract. The action of ultra violet light on the skin changes certain chemical substances into a form of vitamin D so that sunlight as well as nutritional sources of vitamin D prevent rickets. It must be remembered that calcium salts in adequate amounts and in proper balance must also be available in the diet and must be normally absorbed from the intestinal tract. Vitamin D aids in the proper utilization of minerals. It is not a substitute for them.[14]

Vitamin A has also been shown to be essential for the process of bone growth and remodeling. In vitamin A deficiency bone growth ceases and with greatly excessive doses of vitamin A, bone remodeling and the action of osteoclasts is so accelerated that spontaneous fractures, including fractures of the alveolar process, occur.[15]

EFFECT OF HORMONES ON BONE

Next to be considered is the effect of hormones on bony structures. The influence of the parathyroid glands is most clearly established and best known. With excessive function (hyperparathyroidism), the blood serum calcium rises and the serum phosphorous drops. There is a greatly increased loss of calcium through the urine and osteo-

clastic destruction of bone occurs throughout the skeleton including the alveolar process (osteitis fibrosa cystic generalisata).

With excessive secretion of the thyroid gland the activity of all cells is increased. This leads to an increased demand for nutriment including greater requirements for vitamins and minerals. The loss of calcium in hyperthyroidism may be very great and, as Goldman[16] has shown, rarefaction of the bone including the alveolar process may be striking.

The relation of insulin, the secretion of the islands of Langerhans, to diabetes has been thoroughly reviewed by Glickman.[17] Disturbed carbohydrate metabolism is well known to predispose to infectious disease. While there are no specific differences between periodontal disease in diabetics and in non-diabetics, it is well established that diabetics are particularly liable to periodontal disease and that periodontal disease tends to progress rapidly in such individuals.

The next subject is one which is developing very rapidly and in which the chemistry and physiology is extraordinarily complex. It involves the fat soluble steroid compounds found in the gonads and in the adrenal cortex. Fuller Albright[18] said in a recent address that the old aphorism "if you know syphilis, you know internal medicine" might well be changed to "if you know the adrenal cortex, you know internal medicine." Albright continued by saying that he did not know the adrenal cortex well enough to make it the subject of his lecture so you may judge the complexity of the effects of the secretions of this gland. Albright discusses bone rarefaction or osteoporosis in this regard.

Albright defines osteoporosis as a decrease in the quantity of bone where the disturbance is a failure of the osteoblasts to lay down bone matrix. He discusses three factors which influence osteoblastic activity: a) steroidal hormones, b) mechanical stress and strains, and c) nitrogenous building blocks. The influence of stress and strain, that is, functional demands on the alveolar bone and periosteum, is well known to periodontists. Since these structures are built by cells and consist of protein compounds it is clear that the raw materials must be available—the building blocks.

Albright discusses the steroids as they affect bone matrix in two groups, those which stimulate (anabolic) and those which inhibit cell activity (antianabolic).

The anabolic steroids are estrogens and androgens. They are derived from the ovaries and testes and also from the adrenal cortex. The steroid of the adrenal cortex which stimulates bone formation is called nitrogen or "N" hormone.

The type of steroid which inhibits bone formation is derived from the adrenal cortex also and is called the sugar or "S" hormone. Osteoporosis commonly occurs after the menopause. It may be explained as an atrophy of disuse and a loss of the bone stimulating effect of the estrogens, often complicated by a deficient dietary.

At a greater age, usually around 70, the steroids of the "N" hormone type also fail and Albright refers to this as the "adrenopause." It affects both males and females.

The "S" hormone of the adrenal gland is normally produced at a constant level throughout life.

A financial analogy may possibly be helpful. The bone matrix may be compared to our bank balance. We have certain fixed expenses for fundamental needs, such as food and shelter. These expenses go on whether or not we are earning an income. These expenses may be compared to the adrenal cortical "S" hormone.

The estrogens, androgens and "N" hormone of the adrenal cortex may be compared to our earning ability. As long as that is normal our fixed expenses can be taken care of with an adequate margin of reserve.

As might be anticipated, there are situations in which the "S" hormone is pro-

duced in abnormally great amounts and leads to osteoporosis. One of these conditions is Cushing's syndrome which he described as the result of an adenoma of the basophil cells of the pituitary gland but which is now known to be primarily associated with an adenoma of the adrenal cortex. The other is Selye's adaptation syndrome.[19] This is a reaction to injury or infection, the alarm reaction, during which lymphoid tissue breaks down and releases immune globulin, the burning of sugar is interfered with and glycogen is deposited in the liver.

It is obvious that the subject of osteoporosis is of fundamental importance to periodontists. It is interesting to note that Gottlieb's patient with systemic alveolar bone atrophy was a young man, 21 years of age who died of pneumonia in Vienna after World War I. This patient certainly had had an inadequate protein and probably vitamin intake, and in the light of our present knowledge was in Selye's stage of exhaustion due to severe infection with the release of "S" hormone from the adrenal cortex and resultant osteoporosis affecting the alveolar bone as well as other parts of the skeleton.

The pituitary has a direct effect on bone growth and in acromegaly produces enlargement of the mandible even in the adult. It regulates all other glands of internal secretion and in turn its activity is affected by their function in a sort of thermostatic control. When the pituitary is destroyed rapid aging and death occurs (pituitary cachexia). Loss of the teeth by periodontal disease is a part of this clinical picture and the effect of hypophysectomy on the periodontal tissues of experimental animals has been ably described by Schour.[20]

SUMMARY AND CONCLUSIONS

Periodontal disease, like all other disease processes, is the end result of a combination of adverse local and systemic factors. Local factors probably in all instances are the immediate cause of the disease. The reactions of the tissues which tend to prevent invasion by bacteria or to protect against mechanical injury are always dependent for their effectiveness upon systemic factors. Vitamins and hormones have been used as examples of experimental evidence which help to make clear the influence of systemic factors on periodontal disease.

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A Method for Post-Gingivectomy Pack Stabilization*

By JOHN S. MCKENZIE, D.D.S., *Miami, Florida*

SINCE the classic description of "Pyorrhea" by John Riggs[1] in 1878, a wide variety of techniques for the treatment of periodontal diseases have been devised. Gingivectomy, to eradicate the periodontal pocket, was advanced by Bourdet[2] in 1757, and has been further modified by Kirkland,[3] Crane-Kaplan,[4] Ward,[5] A. D. Black,[6] Merrifield,[7] Lundquist,[8] and others since that date.

Numerous surgical packs or dressings have been offered as a protective covering of the wound following gingivectomy. These include Wondrpak,[9] zinc oxide and eugenol,[10] paraffin and cocoa butter, and pure white beeswax.[11]

However, since the advent of the surgical dressing, none have been without shortcomings, the most serious being instability and poor esthetics. Instability necessitated frequent unscheduled visits to the periodontist for dressing replacement during the initial healing period, and encouraged undesirable granulation tissue formation. For the more fastidious patients, and the patients forced to continue their duties before the public, poor esthetics were of major concern.

In 1945, Orban[12] described a removable acrylic veneer to be worn over upper buccal and labial gingival areas AFTER healing of deep periodontal surgery to mask unsightly root surfaces. This was intended solely as an esthetic aid.

This preliminary report describes a continuous acrylic splint, covering the dressing labially, buccally and lingually on either arch, used DURING the healing phase, not only for esthetics, but for the far more important objectives of stabilization of the pack and promotion of better healing.

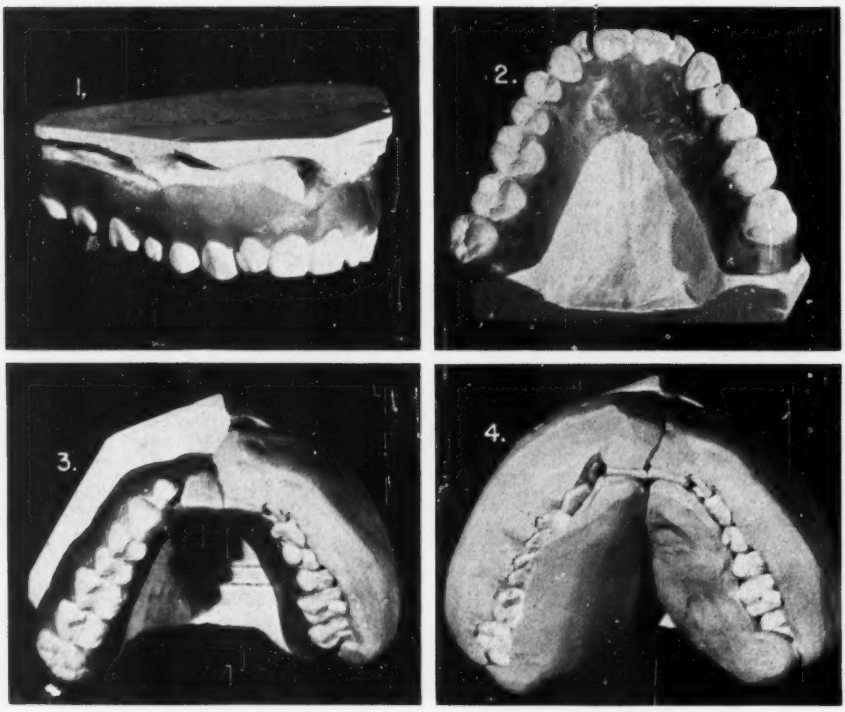
*A Preliminary Report.

MATERIALS AND METHODS

At an appointment prior to gingivectomy, an algenate impression is taken of the arch or arches on which gingivectomy is to be performed, and stone models made. Two thicknesses of 22 gauge casting wax are adapted over the gingivae on the cast to a height several millimeters apically to the depth of the existing periodontal pockets. The wax is carefully carved to the curvature of the teeth necks, and well adapted into the interdental spaces. This is performed in a continuous adaptation labially, lingually, and buccally in cases of complete gingivectomy. (Fig. 1-2) Variations to suit the needs of the individual case can be made where only groups of teeth are to be operated. Separating media is then applied over the cast. (Tincture of green soap works well as a separator). First, the model with adapted wax is rinsed in plain water, then covered with tincture of green soap, and again rinsed with water.

In reproducing the wax pattern in acrylic, the waxed model may be flasked in a denture flask, the wax eliminated, acrylic packed and cured in the conventional manner, or a much simpler method utilized as follows:

A sectional stone core is made over the wax by covering the labial and buccal area of the cast, from the midline to an area distal to the last posterior teeth, with dental stone to a thickness of approximately 2 cm. This is allowed to harden, and is carefully separated and trimmed at each end to eliminate undercuts, replaced on the model, (Fig. 3) separating media applied over both ends of core section, and a similar section made over the opposite labial and buccal portion of the cast in a like manner. The procedure is repeated lingually, extending each section of the core from midline to the distal of the last tooth, so that all wax is completely covered by four sections of core. (Fig. 4) All sections



Figures 1 & 2. Showing adaptation of wax to cast prior to construction of core sections. Figure 3. Labial and buccal core section completed on one side of the cast. Figure 4. Showing all core sections completed.

are removed, and wax pattern removed and discarded.

The cast and core sections are boiled to remove separating media and to cleanse. While the model is still warm it is painted with liquid plastic* to facilitate separation of cured splint from cast. (Fig. 5) After the cast is dry, a thin mix of self-curing, gum-colored acrylic** is applied over the area covered by the first of the sectional cores. (Fig. 6) This core section is then firmly pressed into place over the acrylic, and allowed to cure for fifteen minutes. (A thin sheet of wet cellophane between the acrylic and core section as a separator produces a gloss that needs little polishing.) (Fig. 7) The core and cellophane are then removed, and the operation is repeated with each succeeding section until the entire splint is cured. (Fig. 8)

The core is discarded, and all thin excesses of acrylic on the margin of the splint are trimmed off with a bur or stone and the splint separated behind the posterior tooth on either side with a separating disc. The labial and buccal portion is then removed in one section, the lingual portion in another. Festooning and rough finishing are completed with a flame-shaped vulcanite bur. (Fig. 9)

The splint is replaced on model, and final polishing completed with brush and cloth

*"Plas-Cote" is used by the author, but any similar preparation can probably be utilized with equal success.

**Getz Plastic Model Material works satisfactorily, however, it is not recommended over any other similar gum-colored, self-curing acrylic.

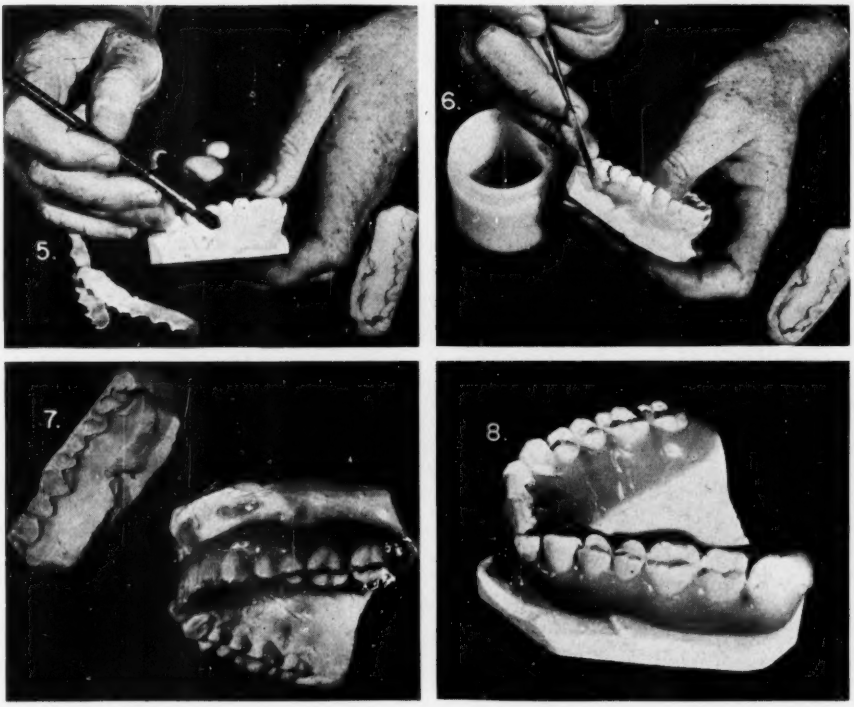


Figure 5. Wax pattern removed, and liquid plastic being applied to cast with camel-hair brush. Figure 6. Thin gum colored, self curing plastic is applied over area covered by labial and buccal portion of core. Wet cellophane is kept in readiness. Figure 7. Plastic pressed and cured. Illustration show core removed, and cellophane still in place. Note gloss obtained. Figure 8. Entire splint is cured. Core sections and cellophane removed.

wheels on a slow-running lathe, using pumice and chalk. (In all cases, splint should be replaced on cast for all polishing procedures to prevent breakage and warpage.)

The operator may now proceed with gingivectomy, utilizing the technique of choice, using slow-setting dressing applied in the usual manner. (The author has found Kirkland-Kaiser dressing easy to manipulate and very satisfactory under splints.) Apply splint by inserting labial and buccal portion first, exerting only enough pressure for close adaptation. Excess dressing is then trimmed with curette or any suitable instrument. (Fig. 10)

The lingual portion is applied in a similar manner. As application is made, firm pressure is maintained on previously applied buccal and labial splint to prevent dislodgment by forcing dressing interdentally. Excess dressing is then removed around lingual portion of splint.

Retention of the splint is obtained by close adaptation of the acrylic to the tissues and dressing, curvature of the dental arch, and by utilization of finger-like interdental projections, similar to those used on so-called "horse shoe" partials.

SELECTION OF PATIENTS AND OBSERVATIONS

Twenty-six patients needing gingivectomy were selected at random from the author's private practice, with no distinc-

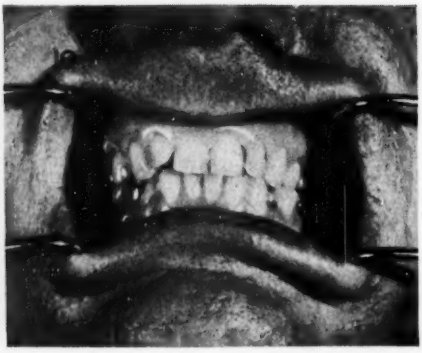


Figure 9. Splint roughly finished with flame shaped vulcanite bur. Figure 10. Finished upper split inserted in mouth.

tion made as to age, sex, or severity of the periodontal breakdown.

On eight of these cases, protective acrylic splints were applied over Kirkland-Kaiser dressing on the upper arch, and Kirkland-Kaiser dressing alone was used on the lower arch. In every instance, patients were forced to return to the office from two to six times during the fourteen day protective period for dressing replacement on the lower arch. In no case was there dislodgment of the upper splint, nor was any adjustment necessary.

In eighteen cases, complete splints were applied, and no dietary change recommended. In no instance was there dislodgment of splint, nor complaint of discomfort. On the other hand, every patient expressed surprise at the comfort and pleasing esthetics resulting.

In six cases, splint and dressing were removed on one arch in eight days, and allowed to remain fourteen days on the opposite arch. In every case, healing was cleaner, with a noticeable absence of granulation tissue in eight days, contrasted with the arch using fourteen day dressings, which slowed visible down-growth of granulation tissue in each instance.

DISCUSSION

The author feels that the stabilizing splint is practical in its application, and fills a much-needed place in the field of periodontal treatment. The technique of construction is a simple one which can be easily learned by the dental assistant.

Esthetics are of major concern to both sexes, and any improvement during the healing period is welcomed by the patient. Lost time and inconvenience suffered by the operator and patient in replacement of lost dressing are eliminated.

SUMMARY AND CONCLUSIONS

1. The chief complaints to immediate post-operative care after gingivectomy have been the lack of stabilization of dressings and poor esthetics. The author has devised an acrylic, gum-colored veneer splint for stabilization of dressing and esthetics during post-operative healing.

2. Construction and application of splint is simple, requires little time, and a minimum of materials and equipment. It can be varied in design to include complete mouths, or groups of teeth.

3. Neither dislodgment nor discomfort has been noted in twenty-six completed cases, and no adjustment was necessary in any case.

4. Esthetics were good in all cases.

5. Clinical healing was cleaner where

splint and dressing were removed on one arch in eight days, than in the opposite arch on which they were allowed to remain fourteen days.

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Relation of Malnutrition, Endemic Dental Fluorosis and Oral Hygiene to the Prevalence and Severity of Gingivitis

By MAURY MASSLER, D.D.S., M.S.* AND ISAAC SCHOUR, D.D.S., PH.D.**

INTRODUCTION

THE purpose of this investigation was to survey the prevalence and severity of gingivitis in two Italian cities, Campagnano di Roma and Quarto, which suffered from non-endemic dental fluorosis. Ricci (1933) conducted a dental investigation on 400 inhabitants of Campagnano di Roma. He did not report any data on gingival disease, but stated that there appeared to be a predisposition to periodontal alterations. The fact that the inhabitants of these cities also suffered from different degrees of malnutrition and showed different degrees of neglect in oral hygiene offered the opportunity to study the possible role of these factors in the etiology of gingival disease.

While the literature contains many reports and reviews (Schour and Massler, 1945) on the possible relation between malnutrition and other factors and gingival disease, the conclusions have been based for the most part on clinical observations and gross statistical comparisons.

An effort was therefore made to analyze statistically the relative significance of endemic dental fluorosis, malnutrition and oral hygiene in the production of gingival disease on an individual basis rather than on the basis of gross group comparisons.

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TABLE 1.

Prevalence and Degree of Gingivitis in Campagnano Di Roma and in Quarto

Age Groups (in years)	Number of Persons Examined	Per Cent of Persons				Average Degree of Gingivitis per Person**
		No Gingivitis (0)*	With Gingivitis			
			Mild (1)*	Moderate (2)*	Severe (3)*	
Campagnano di Roma						
6-10	99	38.4	54.5	7.1	0.0	0.69
11-15	123	4.1	77.2	18.7	0.0	1.15
16-20	79	6.3	59.5	30.4	3.8	1.32
21-30	135	3.0	54.1	34.0	8.9	1.49
31-40	87	1.2	37.9	42.5	18.4	1.78
41-50	54	0.0	22.2	35.2	42.6	2.20
51-60	33	0.0	15.1	39.4	45.5	2.30
	610					
Quarto						
6-10	185	24.3	61.7	12.4	1.6	0.91
11-15	92	19.6	58.7	18.5	3.2	1.05
16-20	23	13.0	69.7	13.0	4.3	1.09
21-30	21	9.5	38.0	47.7	4.8	1.48
31-40	11	0.0	36.4	36.4	27.2	1.91
	332					

*Numerical values arbitrarily assigned in order to compute the average degree of gingivitis per person.
**Degree 0 to 1 indicates predominance of papillary gingivitis
Degree 1 to 2 indicates predominance of marginal gingivitis
Degree 2 to 3 indicates predominance of attached gingivitis (periodontitis)

METHOD AND MATERIAL

The gingivae were examined for the prevalence, degree and type of gingivitis in 610 individuals 6 to 60 years of age in Campagnano di Roma and in 332 individuals 6 to 40 years of age in Quarto (Table 1).

Gingivitis was recorded as being absent, mild, moderate, or severe. In addition the gingivitis was recorded in terms of acute and chronic types. The degree of gingivitis per person was estimated by assigning the numerical values of 0 for no gingivitis, 1 for mild, 2 for moderate, and 3 for severe gingivitis as previously described by Schour and Massler (1947). Data obtained in a previous study on the prevalence and degree of gingivitis in 1588 individuals residing in Naples (a non-endemic but malnourished area) were used as control data in this study (Schour and Massler, 1947). The average degree of gingivitis was obtained by dividing the sum of the numerical values assigned to the individuals in each age group by the number of people in the group.

For details as to the nutritional status and the index of fluorosis in Campagnano di Roma and Quarto see Massler and Schour, 1951. Campagnano di Roma was a relatively fairly well nourished community with a fluoride content in the water supply of 3.5 p.p.m. Quarto, in contrast, was a war devastated area whose inhabitants suffered from severe malnutrition. The fluoride content of the village water

well was 1.3 p.p.m. In both communities, mottling of the enamel was prominent.

Naples was, at the time of this investigation (November, 1945, to February, 1946), just recovering from the effects of World War II. The nutritional level was poor but better than that of Quarto although not as high as in Campagnano di Roma which had escaped the ravages of war by being located high on a mountain top off the main road from Naples to Rome. The fluoride content of the water was 0.2 p.p.m. and no mottling of the teeth was evident even on close examination.

Physical Status. Physical status was measured on the basis of height-weight relation shown on the Wetzel Grid and employed to indicate also the nutritional status.

Oral Hygiene. The degree of oral hygiene was recorded in terms of fair, medium and poor.

Coefficients of Association. Yule's modification of Pearson's coefficient of association was employed to determine statistically the possible association between gingivitis and mottling, oral hygiene and physical status.

FINDINGS

Prevalence and Degree of Gingivitis in Campagnano di Roma. Approximately 62% of the children 6 to 10 years of age showed some degree of gingivitis. The prevalence of gingivitis increased rapidly to 96% in the 11 to 15 year olds and was 100% in those 41 years or older (Table 1).

The average degree of gingivitis per person rose from 0.69 in the 6 to 10 year old group to 1.78 in the 31 to 40 year old group and 2.3 in the 51 to 60 group (Table 1).

Prevalence and Degree of Gingivitis in Quarto. In Quarto, approximately 76% of the children 6 to 10 years of age showed some degree of gingivitis. The prevalence increased to 100% in those 31 years or older (Table 1).

TABLE 2.

Comparison of Gingivitis at Different Ages In Naples, Quarto and Campagnano Di Roma

Age Groups (in years)	*Naples***	*Quarto*	*Campagnano*
A. Per Cent of Persons with Gingivitis			
6-10	54.0	75.7	61.6
11-15	69.6	80.4	95.9
16-20		87.0	93.7
21-30	84.6	90.5	97.0
31-40	92.7	100.0	98.8
41-50	97.2		100.0
51-60	98.0		100.0
*B. Average Degree of Gingivitis Per Person**			
6-10	0.69	0.91	0.69
11-15	0.81	1.05	1.15
16-20		1.09	1.32
21-30	1.53	1.48	1.49
31-40	1.65	1.91	1.78
41-50	1.95		2.20
51-60	2.46		2.30
*C. Average Degree of Acute Gingivitis Per Person**			
6-10	0.69	0.30	0.40
11-15	0.84	0.42	0.83
16-20	0.69	0.44	0.95
21-30	1.11	0.65	1.19
31-40	1.34	0.82	1.54
41-50	1.56		1.48
51-60	1.75		2.55
*D. Average Degree of Chronic Gingivitis Per Person**			
6-10	0.45	0.90	0.54
11-15	0.58	1.05	0.94
16-20	0.50	1.00	1.10
21-30	1.04	1.43	1.23
31-40	1.53	2.18	1.52
41-50	1.95		2.28
51-60	2.53		2.74
Fluorine Content of Water Supply	0.2 p.p.m.	1.3 p.p.m.	3.5 p.p.m.
Nutritional Status of Population	Intermediate	Very Poor	Fair

*Computed as in Table 1
**From Schour and Massler, 1947

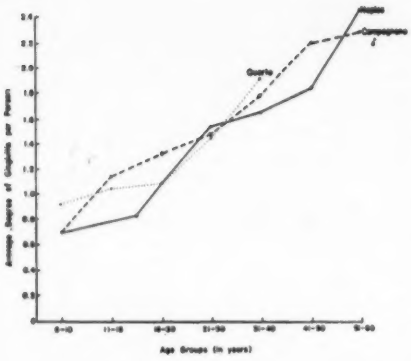

Fig. 1. Comparison of Average Degree of Chronic Gingivitis per Person at Different Ages in Naples, Quarto and Campagnano di Roma.

The average degree of gingivitis per person rose from 0.91 in the 6 to 10 group to 1.91 in the 31 to 40 group (Table 1).

Comparison of Prevalence of Gingivitis in Naples, Quarto and Campagnano di Roma. Table 2A shows that the prevalence of gingivitis (number of persons affected) was somewhat lower in Naples than in Campagnano di Roma or Quarto. The prevalence of gingivitis in the latter two areas was about the same, except for a somewhat higher prevalence in the 6 to 10 group living in Quarto.

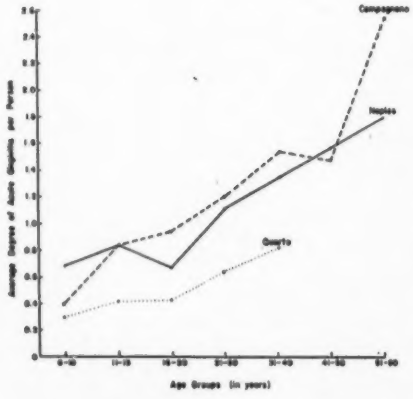

Fig. 2. Comparison of Average Degree of Acute Gingivitis per Person at Different Ages in Naples, Quarto and Campagnano di Roma.

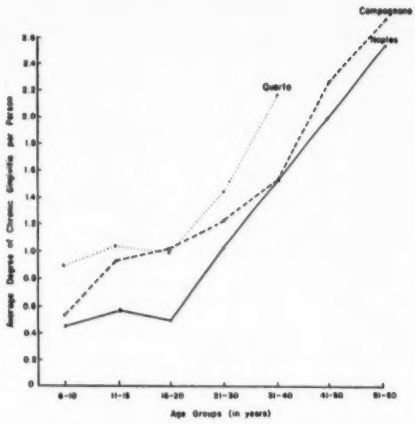

Fig. 3. Comparison of Average Degree of Gingivitis per Person at Different Ages in Naples, Quarto and Campagnano di Roma.

Comparison of Average Degree of Gingivitis in Naples, Quarto and Campagnano di Roma. The average degree of gingivitis was approximately the same in all three areas (Table 2B; Fig. 1).

Acute Gingivitis. The total average degree of acute gingivitis for the individuals 6 to 40 years of age was 0.52 for Quarto, 0.93 for Naples and 0.98 for Campagnano di Roma. Likewise the average degree of *acute* gingivitis was lower in Quarto than in Naples and Campagnano di Roma at all age levels examined (Table 2C; Fig. 2). Statistical testing showed that the lowered incidence of acute gingivitis in Quarto was significant.

Chronic Gingivitis. The total average degree of chronic gingivitis for the individuals 6 to 40 years of age was 1.31 for Quarto, 0.82 for Naples and 1.06 for Campagnano di Roma. Table 2D shows that the average degree of chronic gingivitis was higher in Quarto than in Naples and Campagnano di Roma at all age groups studied except 16 to 20 years. Figure 3 indicates these differences in graphic form. Statistical

TABLE 3

Relation of Degree of Chronic Gingivitis to Degree of Endemic Dental Fluorosis in Children Aged 6 to 14 Years Residing in Campagnano Di Roma and in Quarto

		Campagnano di Roma				*C=0.44*
		Degree of Chronic Gingivitis				
		None	*Mild*	*Moderate*	*Severe*	
Degree of Endemic Dental Fluorosis	None	6	1	0	0	7
	Very Mild	14	5	0	0	19
	Mild	35	60	3	0	98
	Moderate	15	68	8	0	91
	Moderately Severe	1	3	3	0	7
	Severe	0	0	0	0	0
		71	137	14	0	222
		Quarto				*C=0.29*
Degree of Endemic Dental Fluorosis	None	37	68	12	1	119
	Very Mild	16	50	14	4	84
	Mild	7	22	6	0	35
	Moderate	3	16	2	1	22
	Moderately Severe	2	8	1	0	11
	Severe	0	0	1	0	1
		65	164	37	6	273

C=Coefficient of Association.

testing showed that the higher degree of chronic gingivitis in Quarto was significant.

DISCUSSION

A comparison of the total gingivitis experience in the non-endemic area of Naples and in the two endemic areas (Campagnano di Roma and Quarto) indicated that there probably is no strong correlation between gingivitis and endemic dental fluorosis. The data did suggest that there might be a stronger relationship between nutritional status and gingivitis since Quarto with the poorest nutrition showed less acute but more chronic gingivitis than the two better nourished areas—although the difference was not marked and of borderline significance. It was therefore decided to test the association between nutritional status (as determined in each person by means of the Wetzel Grid) and gingivitis and also between gingivitis and mottling on an *individual* basis in order to discover any possible association not revealed by the gross comparisons. Since oral hygiene (degree of oral cleanliness) is often associated with gingivitis, this factor was also tested.

Relation between Degree of Chronic Gingivitis and Degree of Mottling. The fluoride concentration of the water was 3.5 p.p.m. in Campagnano di Roma and 1.3 p.p.m. in Quarto. The index of dental fluorosis was correspondingly higher in Campagnano (2.52) than in Quarto (1.20).

The coefficient of association between the four degrees of *chronic* gingivitis and six degrees of mottling was found to be 0.44 in Campagnano di Roma and 0.29 in Quarto (Table 3). Thus it appeared that chronic gingivitis was more likely to be

TABLE 4

Relation of Degree of Chronic Gingivitis to Physical Status in Children Aged 6 to 14 Years Residing in Campagnano Di Roma and in Quarto

Physical Status (Wetzel Grid Rating)	*Degree of Chronic Gingivitis*				
	None	*Mild*	*Moderate*	*Severe*	
Campagnano di Roma					*C=0.528*
A4	8	6	0	0	14
A3	2	3	0	0	5
A2	9	15	0	0	24
A1	9	18	0	0	27
M	6	16	0	0	22
B1	2	8	0	0	10
B2	1	7	0	0	8
B3	2	1	0	0	3
	39	74	0	0	113
Quarto					*C=0.626*
A4	4	16	4	0	24
A3	1	19	6	0	26
A2	13	23	9	2	47
A1	15	30	6	1	52
M	5	25	1	2	33
B1	2	7	0	1	10
B2	0	1	0	0	1
	40	121	26	6	193

C=Coefficient of Association.

higher in individuals showing a higher degree of mottling. The association between chronic gingivitis and mottling was higher in Campagnano than in Quarto. The coefficients of association indicate that this relation was more than chance but was not very strong since perfect correlation would occur at 0.9 and no correlation at 0.

Relation of Degree of Chronic Gingivitis to Physical (Nutritional) Status as Measured by the Wetzel Grid Rating. The coefficient of association between the four degrees of chronic gingivitis and physical status was found to be 0.528 in Campagnano di Roma and 0.626 in Quarto (Table 4). The association between nutritional status and chronic gingivitis was therefore much stronger than between mottling and chronic gingivitis. The data show that the severity of the chronic gingivitis was much higher in Quarto than in Campagnano di Roma and that this was related to the nutritional status. The fact that the great percentage of moderate and even severe chronic gingivitis occurred in Quarto while only mild degrees occurred in Campagnano led us to assume that the low nutritional status is at least an aggravating factor in the production of chronic gingivitis.

Relation of Degree of Chronic Gingivitis to Degree of Oral Cleanliness. The coefficient of association for the degree of chronic gingivitis and degree of oral cleanliness was 0.433 in Campagnano di Roma and 0.389 in Quarto (Table 5). Thus the association between oral hygiene and chronic gingivitis was positive but not as strong as between nutritional status and chronic gingivitis. However the relation between oral hygiene and gingivitis was stronger than the relation of endemic dental fluorosis to gingivitis.

TABLE 5

Relation of Degree of Chronic Gingivitis to Degree of Oral Cleanliness in Children Aged 6 to 14 Years Residing in Campagnano Di Roma and in Quarto

		Campagnano di Roma				*C=0.433*
		Degree of Chronic Gingivitis				
		None	*Mild*	*Moderate*	*Severe*	
Degree of Oral Cleanliness	Very Clean	45	67	1	0	113
	Clean	5	25	0	0	30
	Fair	16	37	8	0	61
	Poor	0	5	0	0	5
	Very Poor	3	4	6	0	13
		69	138	15	0	222
		Quarto				*C=0.389*
Degree of Oral Cleanliness	Very Clean	8	4	1	0	13
	Clean	21	30	4	0	55
	Fair	33	101	17	2	153
	Poor	0	23	10	0	33
	Very Poor	3	6	5	4	18
		65	164	37	6	272

C=Coefficient of Association (Max.=0.894)

The above findings suggest that some association existed between endemic dental fluorosis (mottling) and gingivitis. But it must be admitted that this association was not very strong. There was a slightly better association between oral hygiene and gingivitis, but this association was not as great as is generally reported in the literature. On the other hand, the association between nutritional status (as determined by height-weight relation on the Wetzel Grid) and gingivitis was very definite. It was therefore concluded that *nutritional status was more frequently related to gingivitis than either mottling or oral hygiene.*

SUMMARY AND CONCLUSION

The prevalence and severity of gingivitis were investigated in three Italian communities. Two of these communities (Campagnano di Roma and Quarto) were areas of endemic fluorosis. Naples served as a non-endemic control. The nutritional status of the population in these areas varied from very poor in Quarto to fair in Campagnano with Naples intermediate.

It was found that gingivitis was more frequently associated with lowered nutritional status (as judged by height-weight relation on the Wetzel Grid) than with endemic dental fluorosis or even with oral hygiene. Further studies of these relationships are indicated.

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Modified Gingivectomy

By M. D. WOLFSOHN, *Buffalo, New York*

"MODIFIED GINGIVECTOMY" is a form of treatment of periodontitis where all well supported gingivae are left in place but the unsupported thin tissues are removed. The epithelium and all diseased tissues comprising the "soft tissue" side of the pocket are eliminated and the roots polished to a smooth, unscratched surface.

ADVANTAGES

The advantage of "Modified Gingivectomy" in periodontitis simplex is that there is a minimum of gum shrinkage and a lesser amount of sensitive teeth. In periodontitis complex with deep vertical pockets, it is of particular advantage. In this condition it is not uncommon to see pockets six and eight millimeters in depth. It would not be practical or good surgery to cut to the outline of the pockets and remove all the tissues forming the outer wall. It would be disfiguring and hard to keep clean.

A somewhat similar condition is very often present in the interproximal pocket, especially where the bone is high up on one tooth as in the intrabony pocket. It is a logical procedure and surgically correct to remove the pathological contents of these pockets, allow them to fill up with a good blood clot, and then protect them with a pack.

It is assumed that in all cases before treatment is started that a thorough study has been made of the case, the occlusion equilibrated, diet corrected, etc.

ARMAMENTARIUM

There are various methods of attacking these periodontal conditions and still stay within the surgical limits presented in the previously discussed principles. They consist of instrumentation, chemicals, ionization, surgery, and electrosurgery.

Berliner[6] has shown a very delicate knife that can be used to remove the epithelium lining the pocket.

The author's preference is in electrosurgery using either the electrocoagulator or the endothermic needle for the following reasons:

a) It consumes less time

b) There is less bleeding

c) The pocket is sterilized

d) There is no bacterial shower systemically

e) The epithelium lining the pocket and the diseased products are destroyed without removing the supporting periodontium.

Two types of diathermy machines may be used in addition to surgical instruments and scalers, to destroy and remove the epithelium and diseased tissues that are found in the periodontal pocket. One is called the "electrocoagulator"[5] and produces a high frequency current. The working instrument is a silver bi-polar electrode as shown in diagram Fig. 1—A and B. That shown in A can be used to reach most surfaces on all teeth, the exception being the lingual surfaces of the lower molars. For these surfaces, an electrode with the points placed the opposite way as in Fig. 1-B can be used. This point may be readily inserted under the lingual aspect of the molar teeth.

The other type of diathermy machine employed is the short wave unit, and with this apparatus the endothermy or "radio knife" is used as shown in Fig. 2.

TECHNIC

The method of using the bi-polar electrode has been discussed in a previous paper.[5] Briefly the following procedure is used:

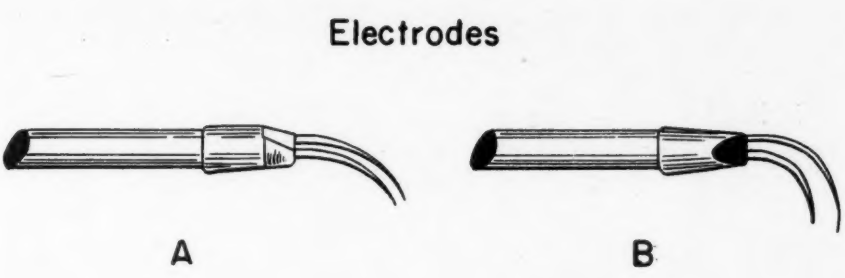

Fig. 1. Bi-Polar electrodes for the electrocoagulator.

The silver points on the electrode are filed to one half their original thickness and made rather blunt near the end. The gums are then painted with an antiseptic, and the area is protected by cotton rolls

Fig. 2. Endothermic needle for short wave diathermy machine.

and suction. The points are inserted to the depth of the pocket and a quick blast of current put through. The points are moved over and another part coagulated. This is repeated until all parts of the pocket are desiccated.

The monopolar or endothermic needle is used differently. The needle is placed in the bottom of the pocket, the foot switch closed and a flash of current is sent through the needle which is moved rapidly in a predetermined path. This is a dangerous instrument and must not be carelessly handled as much damage may be done to the lips, cheek and tongue. They should be well protected by retractors.

The unsupported thin gingival tissue can be totally removed using the regular electrodes or a loop.

The teeth are scaled thoroughly using instruments of choice and the roots polished. It is important that not one piece of calculus be left adherent to the root. The desiccated material is then removed and

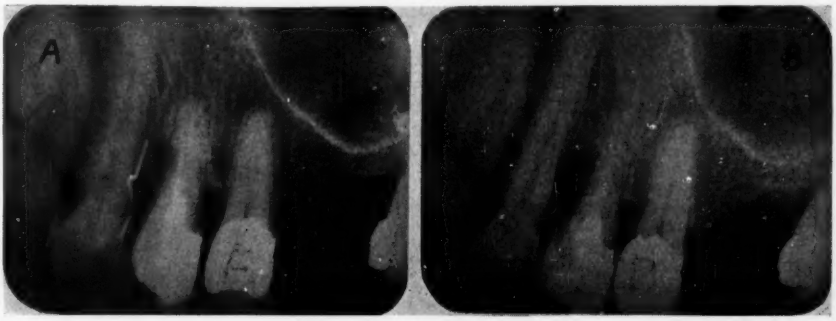

Fig. 3, Case I. (A) Shows a case of periodontal or lateral abscess of a right cuspid with a gutta percha point inserted. The woman fifty years old was treated with endothermic needle (B) Shows same case seven weeks later appears normal.

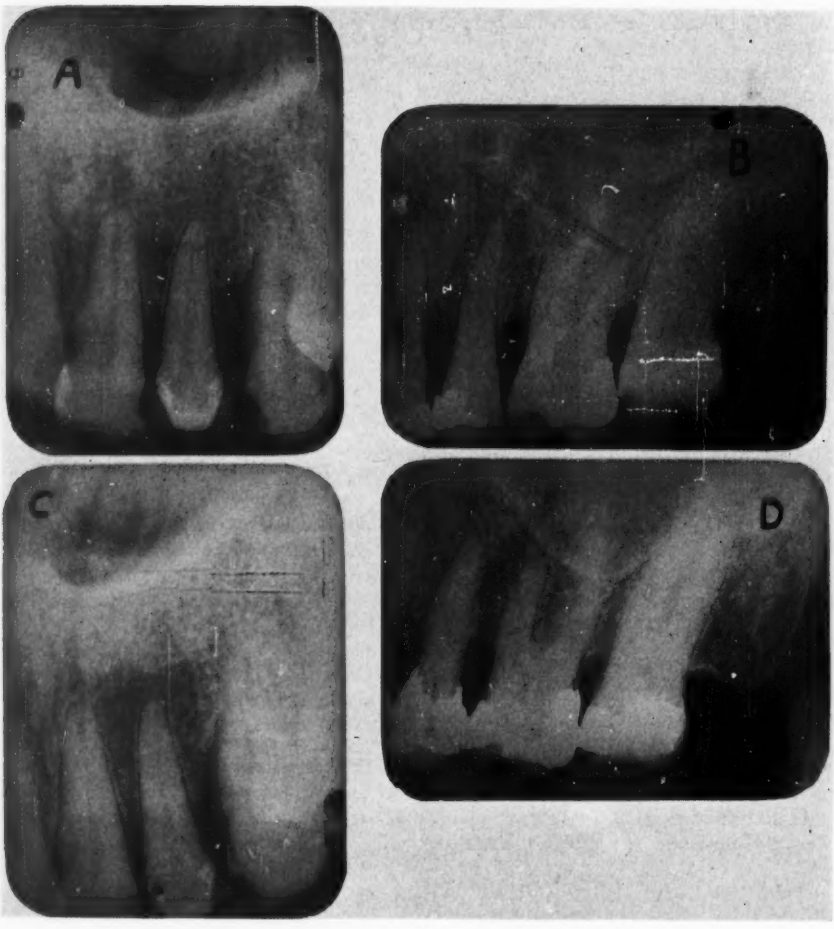

Fig. 4, Case II. (A) (B) Woman forty-five years old showing (1937) periodontitis simplex. Treated with electric-congulation. (C) (D) 1950 shows complete healing of pockets.

the blood clot allowed to form. It is protected by cotton rolls and suction. The gums are forced against the teeth with finger pressure and then packed. Any type of periodontal pack may be used but should be applied rather soft so that the clot will not be displaced.

The pack may be protected by covering it with adhesive tinfoil as shown by Goldman.[3] The pack should be kept in place at least five days, but preferably a week. At that time all the covering material is removed and the tooth area cleaned. Any remaining unhealed pockets are slightly curetted to form a hemorrhage and then repacked for five days. The packing is then removed and the areas flushed out and cleaned. A quarter of the mouth is operated on at a time but this rule can be changed depending upon the type of case and the patient under care.

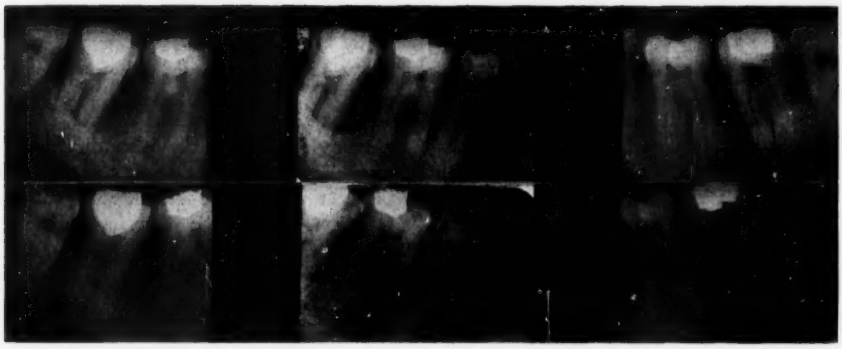

Fig. 5, Case III. Male 50 years old was told that all teeth must be extracted. Teeth were loose with pus exudate from gums. Treated by electrocoagulator, curettage, packing, etc. Teeth are now firm and no pockets present. Upper row 9-10-1948 shows case before treatment. Lower row 2-11-1950 shows same case over two years later.

Pocket eradication is readily attainable regardless of technic, but bone healing is a much slower process. Pocket presence can be easily determined by the use of a periodontal probe or the oxygen insufflation machine, using a twenty-three gauge needle. It is more revealing than the periodontal probe. If there is no ballooning of the tissues and no pain from the needle, it is a positive proof that no pockets exist. Periodontal pockets can be entirely eliminated by electrosurgical methods without removing or resecting the well supported gingival tissues.

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DOCTOR THOMAS B. HARTZELL

Died August 26, 1951

Dr. Thomas B. Hartzell, a pioneer in periodontia in the Midwest, died in Detroit on August 26, 1951, at the age of 85. Dr. Hartzell will be remembered by many because of his energetic endeavors in dentistry. He organized the Periodontia Department at the Dental School of Minnesota in 1904, served as president of the American Dental Association, and was also a past president of the American Academy of Periodontology. In the spring of 1951 he was given a special honorary degree from the University of Minnesota.

Pathological Temporomaxillary Mandibular Relations

By HAROLD G. MORRIS, D.D.S.*

OCCLUSAL trauma, because of a lack of coordinated group thinking, is a most difficult subject to discuss. This situation is being corrected by the acceptance of the pathologists' interpretation of the subject as an inflammatory phenomenon, and by a better understanding of physiological temporomaxillary mandibular relations. A discussion of pathological temporomaxillary mandibular relation may aid in further coordination of thought.

The purpose of this paper is to consider the inflammatory response of occlusal forces and the sequence of reactions at their inception, the premature contact. But first it is necessary to review certain physiological relations.

The mandible at physiological rest, as suggested by Boos, (1), (A, Figure 1) hangs suspended by the temporomandibular investing structures (articular capsule), the suspensory ligaments, and the elevator-depressor muscles. When so suspended, the maxillary dentition and mandibular dentition are separated by a free-way space.

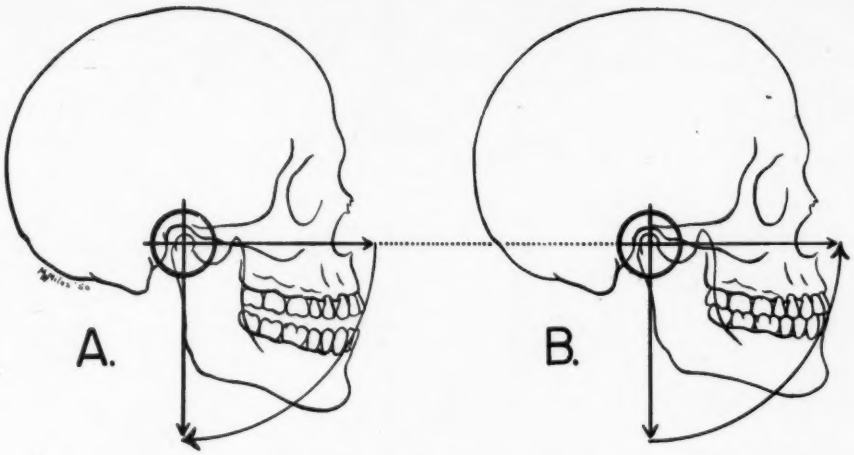
The primary or centric cycle (Figure 1, A and B) of mandibular movement consists of the rotation of the mandible in an arc. The fulcrum of the arc is a horizontal or hinge-axis passing through the heads of the two condyles, and the axis remains physiologically constant in its relation to the articular fossa of the temporal bones. The limits of the centric cycle consist of: 1) physiological rest, 2) an accepted occlusion of the dentition, and 3) a return to physiological rest.

The half-way position (Figure 1, B) or the beginning of return to physiological rest of the centric cycle, is physiological occlusal centric. The centric cycle is the most simple and fundamental of all four mandibular cycles because its half-way point, physiological occlusal centric, is the beginning and end of the other three basic mandibular cycles. The other three cycles are: right lateral, left lateral, and protrusive movements. It is therefore obvious that if occlusal centric is pathological, any and all lateral and protrusive movements made from pathological occlusal centric will also be pathologic.

The premature contact of a supporting cusp or a guiding plane of a maxillary tooth with a guiding plane or a supporting cusp of a mandibular tooth, either arrests the completion of the centric cycle or directs the mandible to acquired or pathological centric. Not only is the dentition involved, but also the hinge-axis is directed from its normal physiological rotating position in its relation to the articular fossa of the temporal bones. The premature contact produces a primary pathological zone. The direction of mandibular movement as produced by muscle effort and the angle of the guiding planes of premature contact either directs the mandible to a position where other teeth are brought into secondary contact with the resultant zones of inflammation, or the hinge-axis is so disturbed that secondary zones of inflammation are created in the temporomandibular structures, suspensory ligaments, elevator-depressor muscles, and adjacent structures. Occasionally the secondary zones of inflammation are more severe and are thus more evident clinically than the primary zones.

By observation of the pathological position of the hinge-axis as directed by the disturbance of the centric cycle, it is possible to classify pathological positions of acquired centric, and by this classification we may more reasonably diagnose, prognose, and treat traumatic occlusion and its ramifications. We may also more satisfactorily discuss and teach the subject. In substance, it forms a basis for interpretation and un-

*Deceased.



THE CENTRIC CYCLE

Figure 1

derstanding. There are fourteen divisions of five classifications of pathological (acquired) mandibular centric:

CLASS I. Retrusive. A-Bilateral. B-Unilateral. Right or Left.

CLASS II. Protusive. A-Bilateral. B-Unilateral. Right or Left.

CLASS III. Reduced Vertical. A-Bilateral. B-Unilateral. Right or Left.

CLASS IV. Increased Vertical. A-Bilateral. B-Unilateral. Right or Left.

CLASS V. Lateral Shift. Unilateral. Right or Left.

CLASS I

TEMPOROMANDIBULAR RETRUSIVE

When a mesial plane or supporting cusp of a mandibular tooth prematurely contacts a maxillary supporting cusp or distal plane, the mandible is directed distally (Fig. 2, Class I). This movement can be bilateral or unilateral, unilateral right or unilateral left, depending on whether the premature contacts are bilateral or unilateral.

The periodontal ramifications are usually negligible. The root formation and root arrangements are better able to withstand mesiodistal forces than lateral forces and adjacent teeth lend support to the offended members. But occasionally contacts which are apparently closed in open mouth examinations are opened under the forces of mastication and so provide avenues for food impaction. The temporomandibular symptoms in the early stages are proprioceptive sense of discomfort which the patient has difficulty in both describing and localizing, and sometimes a temporomandibular joint clicking during mandibular movement. This is followed months or even years later by vague neuralgia-like symptoms, not localized, involving the external auditory canal, temporomandibular area, and, upon occasion, the anterior third of the temporal muscle area. The temporomandibular joint clicking can at this stage be a major complaint. Later, muscle spasms resulting in trismus may be the chief symptom. These muscle spasms and the patient's reaction can so simulate the spasms of tic douloureux that the diagnostician cannot help being tempted to consider trifacial neuralgia and temporomandibular disease

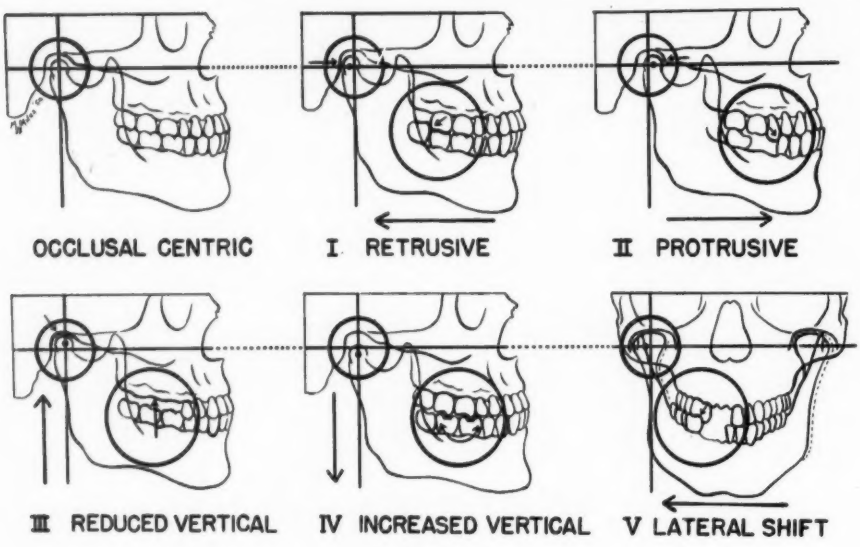


Figure 2

of occlusal disharmony origin, one and the same disease. Perhaps Fulton[3] may have anticipated—consciously or unconsciously—this condition, situation, or observation when, prior to discussing trigeminal neuralgia, he states:

> "Proprioceptive innervation of the striated muscles of the face and orbit has long been a neurological puzzle. Recent studies suggest that the mesencephalic extension of the trigeminal nucleus contains the cells of origin of afferent fibers passing to the muscles of mastication (which also receive motor fibers from the trigeminal nerve). If so this is the one known instance in which cell bodies of afferent neurons are found within the substance of the central nervous system."

The findings in the early phase, other than occlusal disharmony, are a tenderness to palpation of the lateral surface of the condyle, and a lesser degree of tenderness of the distal surface of the condyle upon palpation via the anterior surface of the external auditory meatus. By the application of a stethoscope to the joint area, a tearing or grating sound is registered when compared with the normal joint. The clicking of the joint is definitely registered by the use of the stethoscope. In the later stages of the above, symptoms are exaggerated and tenderness in the temporal muscle area may be diagnosed by palpation.

The causes of this phenomenon are the stretching and injury—a sprain—of the external pterygoid muscle, temporomandibular ligament and temporal muscle with ensuing inflammation. It is usually the inferior fibers of the external pterygoid muscle and the fibers of the temporomandibular ligament and external pterygoid muscle which attach to the head of the condyle. The superior fibers of the external pterygoid which attach to the articular capsule and articular disc are theoretically not injured as the capsule and disc are sufficiently elastic to compensate for the tension, but they may be involved if they are in the zone of inflammation. Fibers within the muscle itself are, on occasion, undoubtedly involved. This cannot be proved by clinical examination or palpa-

tion as the muscle is concealed behind the zygomatic process of the temporal bone and is not available to examination by any known method. The external pterygoid, a depressor muscle, contracts during the elevation of the mandible in accordance with the fact that antagonistic muscles or muscle groups act together, some as movers, the others as stabilizers, thus establishing a muscle balance, as stated by Sicher.[10] The muscle is also subject to the "Stretch Reflex."[3] If a pull is exerted upon the tendon of a muscle by the contraction of an opposing muscle as the result of gravity acting upon the organism or by any number of artificial means, the muscle resists actively the extending force. This active resistance of the muscle is a reflex contraction—the "Stretch Reflex."

The contraction of the external pterygoid as the mandible is directed posteriorly by the occlusal planes and elevated by the elevator muscles, finds the muscle less able to withstand the force of tension. This starts an adverse action within the contracted muscle and something must give way, either muscle fibers or attaching fibers, and a sprain results. The anterior fibers of the temporal muscle are stretched under tension as the coronoid process moves distally. The posterior fibers are not affected. To prove diagnosis of inflammation within one of the external pterygoid muscles, contract the muscle in question by having the patient move the mandible in the opposite lateral cycle. The evidence of pain is ample proof of muscle injury.

Burkett[2] says, the temporomandibular joint "clicking" symptoms frequently given by patients as a complaint is probably clinical evidence of temporomandibular joint subluxation.

Shapiro[9] has demonstrated that the superior fibers of the external pterygoid muscles are attached to the articular capsule and the disc while the inferior fibers of the external pterygoid muscles are attached to the head of the condyle. In lateral mandibular movement the head of that condyle, which is the fulcrum of rotation, rotates on the inferior surfaces of the disc and the opposite condyle remains fixed in its relation to the disc, the disc gliding on the anterior articular eminence. When the jaw is protruded or retracted, motion occurs chiefly in the upper part of the joint with the articular disc gliding forward and backward on the surface of the glenoid fossa.[9] When the attaching fibers of both the temporomandibular ligament and inferior fibers of the external pterygoid have been ruptured and the sprain is sufficiently extensive, the head of the condyle can assume a position posterior to its normal position in relation to the disc—a subluxation. During a forward motion of the subluxation condyle, the superior fibers of the external pterygoid muscle move the disc forward causing it to glide on the anterior eminence. The inferior muscle fibers then advance the posteriorly positioned condyle causing it to slap against the disc with the resultant clicking sound.

Occlusal adjustments to correct Class I malocclusion are made upon primary contacts which are mesio-mandibular planes and disto-maxillary planes of posterior teeth or lingual planes of maxillary incisor teeth. The laws of centric relation and centric adjustment are primary laws and have precedent over the secondary laws of lateral and protrusive movements and adjustments.

CLASS II

TEMPOROMANDIBULAR PROTRUSIVE

When a mandibular distal plane or a supporting cusp prematurely contacts a maxillary supporting cusp or a mesial plane, the mandible moves forward and naturally the hinge-axis moves forward also. As this is a normal mandibular movement simulating the protrusive cycle without the function of the external pterygoid muscles, there is theoretically no temporomandibular pathological involvement and none has been clinically observed by this author.

The pathological movements of Class II

can be bilateral or unilateral, unilateral right or unilateral left, depending on whether the premature contacts are bilateral or unilateral.

Class II traumatism accounts for a high percentage of the mesiodistal pathology of molars and bicuspids. The posterior teeth and their supporting structures are anatomically designed to best accommodate forces parallel to their long axis. But in Class II classification, the forces on mandibular posterior teeth are mesial and on the maxillary dentition they are distal. These forces (one of thrust, the other of tension) compress and thus impair the circulation with the resultant degeneration on the mesial mandibular and distal-maxillary dentition and stretch or create a tension which causes proliferation and degeneration on the distal mandibular and mesial maxillary dentition. They are the most numerous of all forces because the lateral and protrusive cycles begin and end in centric. They are the greatest of all forces because centric forces are the greatest. They are slow crushing forces of long duration (time factor). As facets are cut on the offending members the forces become more crushing; they are greatest in the third molar region because of its proximity to the fulcrum of leverage. Due to increased interproximal and occlusal abrasions these forces increase with time. The repair and proliferative ability of the host must be great to withstand these forces over a long period of time. They are primary traumatic forces.

The distance that the mandible moves in Class II is considerably greater than in Class I. One case of approximately 6/64 of an inch has been observed and 3/64 to 4/64 are not uncommon.

The primary contacts and forces of Class II malocclusion result in and are the cause of secondary contacts which produce other zones of inflammation. As the mandible moves forward and upward to acquired centric supported by the guiding planes and supporting cusps of the posterior teeth, the incisal edges or labial incisal angles of the mandibular incisors contact the lingual planes of the maxillary teeth with a sharp hammer-like blow. A few of nature's responses, in the form of secondary zones of inflammation to the secondary traumatic forces, follow. The forces on the mandibular incisors are to a great extent parallel to the long axis of the tooth, forces which are best accommodated by the periodontal membrane due to their anatomical design. Since the roots are small and saber-like, the apical area receives more of the force than do the horizontal and oblique periodontal fibers. These forces result in chronic inflammation and degeneration of the supporting bone, as is evident in the x-ray picture of osteoporosis or localized rarefication of the bone in the apical area.

Often there is a localized area of condensing osteitis, formative osteitis, sclerosing osteitis, or traumatic hyperplasia, closely-knit bone lamella forming a solid base or foundation for the traumatized teeth, with a thickening of the lamina-dura. When this condition exists, it aids in diagnosis and indicates that the proliferative power of the patient is great. The question arises as to whether the proliferation is a recent and/or still active condition, or the result of previous response that is still clinically evident.

Secondary traumatism results in the semilunar crescent area of resorption and pocket formation on the lingual of the maxillary incisors. The secondary force moves the maxillary incisors labially. The fulcrum of the teeth being below the middle of the root, the greater movement is in the incisal region as related by Kronfeld.[5] The cervical lingual horizontal periodontal fibers are stretched beyond their physiological limits and inflammation ensues. Over a period of time nature tries to compensate for this trauma and the teeth assume and reassume new labial positions. This is a fan-like spread with diastemata appearing between the incisors. In the zone of stimulation of secondary traumatized incisors, the proliferative picture is not infrequently inflammatory and associated

with connective tissue hyperplasia. This hyperplasia is usually more pronounced labially than lingually. The action of the tongue and passage of food over the lingual area either offer sufficient lingual stimulation to aid in the control of the bulk of hyperplasia as compared to labial surfaces, or it is less evident because of the thickness of the tissue. In the youth the hyperplasia has a tendency to be of the inflammatory type, and this secondary traumatization is probably a contributing factor in the adolescent, pubertal, and dilantin hyperplasia. In fact, there is reason to believe that some "so-called" hereditary hyperplasias are in reality trauma-incited. Later in life the connective tissue hyperplasia can often be attributed in part to secondary traumatisms.

Robinson and Kerr[8] recognize excessive thickening, or connective tissue hyperplasia, of the free margin as McCalls' festoons. There is frequently gingival necrosis producing Stillman's cleft.[8] The following observation helps to locate the angle of force associated with the cleft. The Stillman's cleft is usually directly opposite the facet of traumatization and should the cleft at its termination incline either mesially or distally, the facet will still be opposite, either lingual distal or lingual mesial.

Adjustments for Class II protrusive malposition are made on the offending surfaces of the primary contacts, the mandibular distal planes and maxillary mesial planes. Many times a secondary contact induced by the primary contact is observed on the occlusal ridge of the buccal cusp of the maxillary first bicuspid, and the mesio-occlusal plane of the lingual cusp of the maxillary first bicuspid, shifting the mandible laterally. According to the laws of lateral adjustment these are both guiding planes, but the laws of centric supersede and therefore adjustment for centric precedes all other adjustments.

Due to tilting or rotation of the teeth, planes and cusps frequently lose their anatomical identities in occlusal relationships and this must be taken into consideration in occlusal adjustment. The occlusal surface of a mesial-inclined mandibular molar can become a mesial plane; a distal plane of a distal marginal ridge can assume an occlusal plane position.

CLASS III

REDUCED VERTICAL

Posterior teeth, when in physiological occlusal centric relation, resist the forces of tension as applied by the elevator muscles, and thus maintain normal physiological temporomandibular norm. When the resistance offered by the posterior teeth is lost, permitting the anterior teeth to contact excessively, the forces as applied by the elevator muscles reduce the condyle-fossa relationship and the hinge-axis is raised from its physiological position.

The pathological position of Type III can be bilateral or unilateral, unilateral right or unilateral left, depending on whether posterior tooth support is missing on one or both sides of either arch.

Within this classification we can account for a high percentage of temporomandibular disturbance, temporomandibular disease, and the symptoms of Costen's Syndrome.

If the anterior articular eminence of the temporal fossa were non-existent, the temporal fossa area would be a flat plane parallel to the occlusal plane, and if the anterior teeth articulated in an end to end relationship, then the hinge-axis could move up, and up only, closing the vertical dimension within the limits of the compressibility or depressibility of the articular capsule. The anterior articular eminence is, however, an incline plane and the lingual surfaces (guiding planes) of the maxillary incisors are incline planes and in unison they direct the head of the condyle superiorly and posteriorly. The distance of movement is controlled by the longest of these two planes, the rapidity or speed of movement controlled by the plane of great-

est inclination, and the force controlled by the elevator muscles.

Inflammation of the temporal and external pterygoid muscle has been described and interpreted under Class I. In Class III it is usually magnified in both muscles. The articular capsule is in part protected from pathological movement by the superior fibers of the external pterygoid, but the sharp anterior border of the condyle, which has moved up and back, comes in opposition to the articular disc and the articular disc is caught between this margin and the articular eminence during mandibular function.

Sicher[10] reports a flattening of the condyle and articular eminence resulting in resorption and reactive proliferation of the bone, degeneration of parts of the capsule and degeneration of the fibers covering the condyle and tubercle. He terms this condition, traumatic osteo-arthritis deformans. Burket[2] in discussing traumatic disease of the temporomandibular joint (excluding fractures) speaks of acute traumatic temporomandibular arthritis. We may accept either of these terminologies for the advanced stages of the condition. In the primary phases it is simple inflammation, subsequent degeneration, and proliferation over a continued period of time and may result in true osteo-arthritis. Sicher[10] also states that in approximately 20 per cent of all skulls there is a flaw of development resulting in a foramen in the tympanic plate. As Class III thrust of the condyle is directed in this general direction, it is reasonable to assume that should there be inflammation in this region, not only are the tympanic plate, the foramen, and the soft tissue of the foramen involved in this zone of inflammation, but also the acoustic meatus may be included.

Often parotid tissue invades the lateral limits of that area distal to the condyle.[9] Should the invasion be sufficiently deep or the limits of temporomandibular inflammation be sufficiently extensive, parotid tissue can be involved. Burket[2] mentions joint involvement secondary to supportive parotitis. We should give more consideration to pyogenic infection of the parotid gland secondary to inflammation of traumatic temporomandibular joint origin.

The auriculotemporal nerve passes posteriorly and down on the inner surface of the external pterygoid muscle after leaving the foramen ovale and then crosses the posterior border of the neck of the condyle. The posterior border of the neck of the condyle is not necessarily within a zone of inflammation, but the neck can be within such a zone, as it associates with the external pterygoid. The auriculotemporal nerve, a specialized nerve receiving and conducting pain, should receive consideration in temporomandibular pathology as a source of clinical evidence of pain.

The chorda tympani, containing taste fibers for the anterior region of the tongue and secretory fibers for oral glands, anterior lingual, submaxillary, sublingual, etc., passes in a deep narrow grove (petrotympanic fissure) behind the articular capsule and is separated from the capsule by the bony border of the fossa. It is also within the zone of Class III induced inflammation. The stimulation can produce changes in salivation and excessive sensitivity of the tongue and burning, and acid taste which are frequently given by patients as their chief complaint.

We do not pretend that all pathology of the temporomandibular area is trauma-induced, or that if and when trauma is a factor, Class III malposition interpretation will answer all the questions relative to Costen's Syndrome and other vague complaints and symptoms. For example, a furuncle situated on the anterior wall of the external auditory canal can produce symptoms similar to those experienced in temporomandibular disease. The enlargement of the small lymph node just anterior to the temporomandibular joint[2] can be associated with any inflammation in that general area. We should consider that many symptoms are a result of the traumatic-induced inflammation and not of the force itself.

Burket,[2] in discussing the temporomandibular syndrome states, "When the symptoms are present they do not arise until 5 to 8 years after the mutilation of the occlusion." If we associate this observation with the incidence of periodontal bone degeneration in young patients, as recorded in the Gilson chart[4] we will be more alert in both interpretation and diagnosis and be more inclined to practice preventive occlusal adjustment with prevention of inflammation as the goal. If we accept the work of Valy Menkin[6,7] we cannot consider the inflammatory phase of temporomandibular disease as strictly a local condition.

The periodontal results of Class III centric are known to be an overburdening of the remaining teeth (an insufficient number of teeth for equitable distribution of forces). The primary traumatism of anterior teeth is quite similar to secondary traumatism of Class II malcentric, and necessarily the pathological evidence is not dissimilar.

Sensitivity of the necks of anterior teeth, especially anterior maxillary teeth, is often one of the primary symptoms. This can be attributed to trauma-induced hyperemia of the pulp, the inflamed nerve being more receptive to stimuli than a physiologically normal tooth. The observation has been made that not only does the rectification of trauma aid in the reduction of sensitivity but also that the necks of the teeth are less susceptible to caries after correction.

CLASS IV

OPEN VERTICAL—UNDERCLOSURE

Class IV is the man-made traumatism, the dentist-made pathological factor. The centric cycle is interrupted before the completion of the cycle. The premature stop is usually confined to a relatively small area in the bicuspid or molar region, and the functioning muscles attempting to close the cycle cause the mandible to rock in a teeter-totter manner.

The pathological interruption of Class IV can be bilateral or unilateral, unilateral right or unilateral left, depending on the site of interference.

The primary offender is the occlusal surface restorations of posterior teeth, the cusps having been bridged either partially or completely, the occlusal planes not having been carved to original form, leaving angles which are not in harmony with occlusal planes of adjacent teeth and teeth in opposition. This failure of design prevents supporting cusps and guiding planes from functioning in a physiological manner.

One authority teaches that a restoration is either a physiological or a pathological entity, and that a restoration is not just a static creation based on our knowledge of the mechanical arts, but is, rather, a living dynamic entity, a physiological stimulant or a pathological factor. It is the choice of the operator which type of restoration he creates. Frequently the choice is evident as Class IV disturbance of the centric cycle.

Among the oral manifestations are resorption in the bifurcation and trifurcation of molars. The trifurcation pathology is less easy to diagnose as the lingual root is usually superimposed over the area in the x-ray picture. The resorption or degeneration at the apex of bicuspids can be caused by forces of traumatism which are parallel to the long axis of the tooth, the anatomical tooth shape being such that this great force is all apical. Occasionally this degeneration is diagnosed as apical granuloma or, on mandibular bicuspids, as the mental foramen.

Other symptoms are: a constriction, tenseness, fatigue or neuralgia-like sensation of the throat, or tenderness in the general region of the angle of the mandible.

A teeter-totter action of the mandible which alternately increases or decreases the the physiological hinge-axis in its relation to the temporal fossa, also causes a stretching or tension of the suspensory ligaments, sphenomandibular, stylomandibular, and the elevator muscles (buccinator and in-

ternal pterygoid) at the mandibular sling (the region of the angle of the mandible at body and ramus).

Robinson and Kerr[8] report, "Occlusal trauma may be accentuated by bruxism. Equilibration of occlusion frequently eliminates bruxism, thereby eliminating an important traumatic factor." The important traumatic factor or irritant of bruxism is frequently found under Class IV classification.

CLASS V

LATERAL SHIFT OF MANDIBLE

In Class V the horizontal position of the hinge-axis remains constant in its horizontal relation to the temporal fossa but the axis moves laterally or longitudinally right or left, as the case may be.

The movements of Class V are unilateral only, right or left.

This condition exists in cross-bite cases when the buccal planes or buccal cusps of maxillary teeth disturb the centric cycle by prematurely contacting the occlusal planes of buccal cusps of mandibular teeth, or when an occlusal plane of a maxillary lingual cusp prematurely contacts the lingual plane or a mandibular lingual cusp, shifting the entire mandible laterally.

The lateral shift can cause an impingement of the mesial surface of the head of the condyle and the lateral surface of the articular fossa, as an examination of skulls will reveal.

Three cases of lateral mandibular shift have been observed by the author, two of these can be attributed to restorations and a fixed bridge—the guiding planes and supporting cusps were not designed to promote physiological occlusal centric. No temporomandibular symptoms were present, periodontal degeneration was present, both buccal and lingual, in line with the force thrust.

SUMMARY

Occlusal trauma and its ramifications can be best interpreted in terms of temporomaxillary mandibular relations.

Inflammation is the response of periodontal tissues, temporomaxillary-mandibular structures, elevator and depressor muscles, and suspensory ligaments, to trauma of dental origin.

The primary movement of the mandible is the centric cycle which consists of the elevation of the mandible from physiological centric to physiological occlusal centric and its return to physiological centric, the condyle hinge-axis remaining physiologically constant in its relation to the temporal fossa.

Any interference in the path of the centric cycle in the form of premature occlusal contact produced a primary zone of inflammation within the periodontal structures of the offending teeth. The direction of the mandible as directed by the primary contact produces secondary contacts of other teeth and secondary zones of periodontal inflammation, or zones of traumatization within the temporomandibular investing structures, adjacent tissue, elevator and depressor muscles and suspensory ligaments, in other words, a sprain.

Interference with, and the subsequent deviation of, the centric cycle diverts the hinge-axis from its physiological relation to the articular fossa of the temporal bones and by the pathological repositioning of the axis we can classify mandibular excentrics.

The various pathological temporomaxillary mandibular relations can be divided into 5 classes as described above.

The use of the pathological classification of the centric cycle has been an aid to the author in diagnosis, prognosis, and treatment. It is hoped that it may be a basis of understanding relative to the most difficult and least understood of all dental subjects: the disease inflammation as found in the temporomaxillary mandibular structures when caused by disharmony of occlusion.

(Bibliography on page 243)

The Cervical Enamel: Its Physical Characteristics During Curettage

By A. B. RIFFLE, D.D.S., *Rochester, N. Y.*

WHILE curetting the enamel surface, particularly in the mouths of young people, and in the presence of the more active types of gingivitis, one usually encounters a definite crustiness, easily detectable with a sharp instrument. This "crunchy," "gritty" feeling is a distinctly different feeling from the glassy smooth sensation imparted by healthy enamel and is customarily interpreted by the operator as being calculus, unless visible decalcification or actual caries is present. It has been our clinical experience that calculus is rarely present in early gingivitis, while the softened gritty surface of the enamel itself occurs very frequently at all ages, even though it appears normal to the naked eye.

For proof, we have curetted the enamel surface of 500 extracted teeth and we believe we have demonstrated that the enamel, in the cervical third especially, is rarely the hard, smooth, almost impervious substance it is commonly considered to be.

HISTORY

Many men have recognized the presence of this deteriorated surface. Dr. Robert Good,[1] associate to Dr. William Younger, says, "I have seen him remove the entire surface of dead enamel, as he calls it, until he comes to true enamel." Hartzell[2] noted that the enamel at the cemento-enamel junction sometimes ends in an "abrupt, jagged manner." Miller[3] recognized variations in the physical structure of the tooth surface. Black[4] believed there was "so little organic matrix in the enamel that it will not hang together," but disproves his statement by a series of photomicrographs of early enamel caries in which the enamel does "hang together," noting that there is an early softening accompanied by a swelling of the deteriorating tooth structure. Williams[5] clearly demonstrated this and proved these defective surfaces to be commonplace. Head[6] also studied enamel softenings chemically, changing the character of the surface to a sharp lancet without visible change in appearance. Gottlieb,[7,8] Diamond and Weinman,[9] and Weinman, Wessinger, and Reed[10] recognized immature and incompletely calcified enamel. McCall[11] noted that inflammatory exudates could promote superficial decalcification of enamel. Many others have also noted similar changes.[12,13,14,15] (many others)

What actually happens when the enamel is curetted and planed with a sharp instrument seems to have been neglected.

THE STUDY

Five hundred extracted teeth were selected from the offices of three oral surgeons. All teeth were accepted as they appeared in routine sequence, discarding only those where caries, restorations, or mechanical injury rendered them worthless for our purposes. All teeth from each patient were placed in a separate bottle of zephiran chloride solution, having previously ascertained that months in this solution seems to have no effect on the physical characteristics to be studied. It is more satisfactory to work on a wet tooth; even a short drying changes the characteristic feel. On each bottle the surgeon noted the age, the sex, and the reason for extraction. The statistical subdivisions of this information are shown as Table 1.

Each tooth was then photographed on each of its four surfaces, and careful observations of the gross appearance of the enamel recorded, especially in the cervical

TABLE I

Summary of the General Observations on the 500 Extracted Teeth Used in the Study

Total Number of Teeth	500
Total Number of Patients	312
Sex	
Male	184
Female	306
Unknown	10
	500
Tooth	
Incisors	131
Cuspids	66
Bicuspids	114
Molars	176
Deciduous incisors	1
Deciduous cuspids	1
Deciduous molars	11
	500
Age Group	
Unknown	10
1 (0-15 years)	36
2 (16-25 years)	81
3 (26-35 years)	88
4 (36-50 years)	162
5 (51 years and over)	123
	500
Reason for Extraction	
Periodontoclasia	221
Caries	164
Caries and Periodontoclasia	52
Orthodontic	21
Traumatic	3
Impacted (Full and Partial)	10
Prosthetic	9
Unknown	20
	500
Periodontal Classification (Cemento-enamel junction to remaining soft tissue attachment)	
0	62
1 (0 to ¼ cemento-enamel junction to apex)	193
2 (¼ to ½)	135
3 (½ to ¾)	82
4 (over ¾)	28
	500

area. Accurate measurements of the distance from the cemento-enamel junction to the apex were also made on each of the four surfaces, for comparison with similar measurements following curettage.

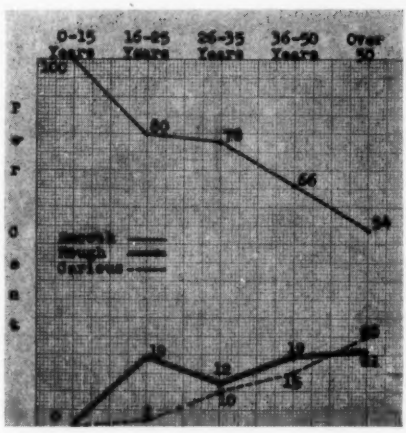
The enamel of each tooth was then curetted with a sharp instrument, carefully recording the degree of surface softening and classifying this as Definite Crust, Slight Crust, or O Crust, curettage being continued until the glassy hard smooth feel we interpret as healthy tooth structure was reached. Early teeth were curetted with standard curets such as Towner's, Gracey's, McCalls, etc., but this soon proved too difficult. Thereafter we used a Kingsley scraper, being satisfied that the end results are the same. Constant resharpening of the instrument was required, particularly just before the completion of the planing operation.

Photographs were then retaken in the identical position as before curettage and the measurements from the cemento-enamel junction to the apex repeated.

THE ENAMEL BEFORE CURETTAGE

The visible characteristics of the enamel surfaces in the cervical third before curettage are recorded in Graph 1. Each tooth was considered to have four surfaces, but no breakdown was attempted to determine the surfaces most frequently showing a noticeable variation from the normal. Thus, if one tooth has three normal surfaces and one surface slightly roughened or carious, it would appear in the totals as 25% deteriorated.

Before 16 years, all teeth appeared macroscopically normal. From then on through life there is a gradual decrease in the frequency of the seeming normal and a corresponding increase in the frequency of the visibly abnormal, except for the period from 26-35 years. During this period there seems to be a slowing of the rapidity with which new changes occur—almost a cessation of activity. This is of interest, for all approaches we have made in the study of the progress of periodontal diseases seem to show this plateau. It is our clinical impression that this is true at the chair—that periodontal symptoms decrease during this age period—but have not correlated sufficient evidence to present it as



Graph 1. The Character of the Cervical Enamel Before Curettage. See text.

a truth. The next paragraph and later articles will show other evidence of this phenomenon.

During the period from 16-25 years, 19% of the enamel surfaces are visibly roughened, which percentage drops to 12% during the period from 26-35 years. (Graph 1—Rough) At first we considered this a chance result, within the limits of probability of error, but as we have watched this characteristic clinically and considered it carefully, we now believe it to be representative of the actual course of events. Normal tooth eruption and its accompanying gingival recession, plus the slowing or cessation of the activity of the periodontal lesions as mentioned above, exposes the cervical enamel. Some surfaces remain little changed as a dull, stained, lifeless appearing tooth; on others the usual frictions of tongue, cheek, mastication, toothbrushing, etc., slowly wears the roughness away; on still others the area becomes carious. Notice that the total of both roughened and carious surfaces is about the same in both age groups, as if new surface deterioration had ceased, but caries had invaded some of those already present.

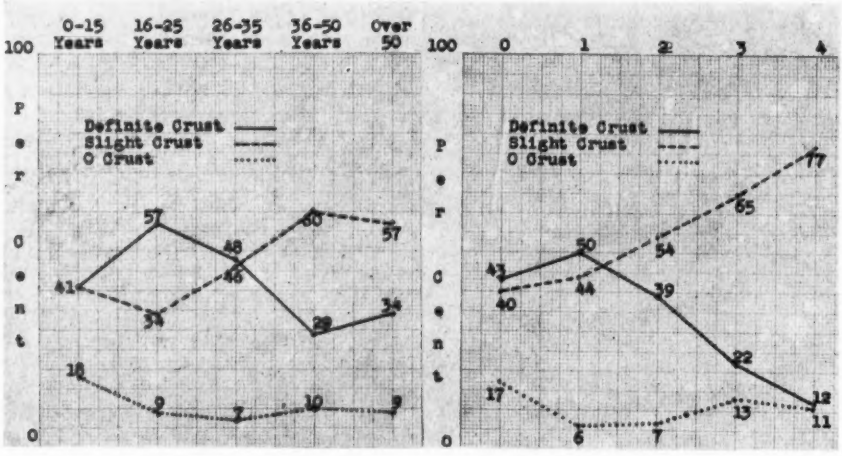
Caries of the enamel in the cervical area—not cemento-enamel junction caries (Graph 1—Carious)—is not common until after 25 years, and becomes progressively more common through life, 25% of all surfaces being affected in the teeth from mouths over 50 years of age. Some of this increased incidence of caries in later life is undoubtedly an incisal/occlusal extension of caries of cemento-enamel junction origin, though we attempted to separate these and not record them in this series.

To correctly interpret Graph 1, the totals of both roughness and caries are undoubtedly the important thing, with the conclusion that *visible deteriorations of the surface of the enamel in the cervical third increase in incidence in ratio to advancing years, except for the period from 26 to 35 years.*

ENAMEL SURFACE DETERIORATIONS

During the process of scraping the cervical enamel with a sharp instrument, one is surprised at the frequency with which a definite crusty surface is encountered, in contradistinction to the smooth, glassy feel found on other teeth, or even on the incisal/occlusal third of the same tooth. The first feeling of this crust may be a definite softness like the feeling of softened dentin after caries has been removed, or it may be a barely detectable "grittiness." If the feeling is one of softness in the beginning, one may expect a considerable loss of structure, each stroke feeling harder, until finally the last fine "sandiness" gives way to the hard, smooth, glassy feel of healthy tooth structure. If the feeling in the beginning is already one of a very hard, fine roughness, one may expect a very superficial loss before this hard smooth surface is attained.

On some the crust is entirely missing (O Crust); on some there is only the most minute loss of surface substance (Slight Crust); on others there is a macroscopically visible loss of surface, or even a complete loss of the enamel before a hard smooth



Graph 2. The Enamel Surface Crust, showing the per cent of surfaces involved and the frequency of incidence of the various degrees of crustiness. Each tooth was considered to have four surfaces, so the percentages are of 2,000 surfaces, not 500 teeth.
A. The teeth are subdivided into age groups.
B. The teeth are subdivided into an arbitrary classification in which at O, the remaining attached fibers of the periodontal ligament are attached at the cemento-enamel junction; at **1**, apical to the cemento-enamel junction but less than ¼ the distance to the apex; at **2**, more than ¼ but less than ½; at **3**, more than ½ but less than ¾; and at **4**, more than ¾.

surface is reached (Definite Crust). Many have one or more surfaces with no crustiness, and marked crustiness on one or more of the other surfaces.

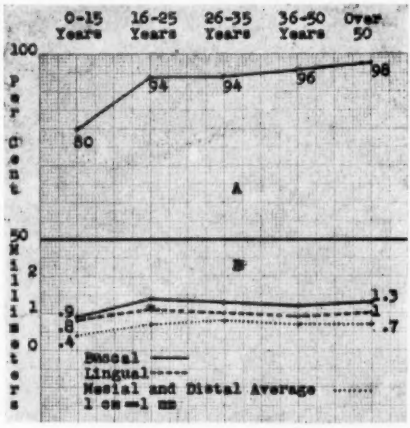
Definite Crust, Slight Crust, and O Crust were the only distinctions we attempted to record statistically.

Graph 2 depicts our findings. The teeth were subdivided first into age groups and the findings recorded (A), and then redivided into an arbitrary classification of the depth of the pathologic crevice as represented by the clinging fibers of the periodontal membrane (B). The legend describes this classification.

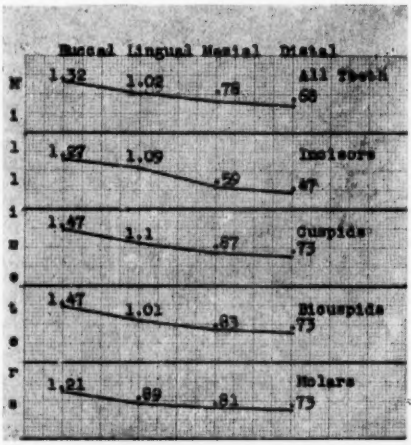
Before 16 years (Graph 2-A) we find 82% with either a Slight or a Definite Crust, and only 18% glassy hard and smooth to the first strokes of the curet. These could all be normal, for Gottlieb[8] tells us that "under normal conditions the thin outer surface layer of the enamel matrix does not mature," recognizing, however, that on some, complete calcification does take place. Weinman, Wessinger, and Reed[10] also have shown that erupting teeth may be matured at the incisal/occlusal and immature at the cervical.

The normal sequence of events is for this immature cervical enamel to mature[9] and failure of maturation is pathology.[16] Therefore, though we might consider the Slight and Definite Crusts of early life as the normal, surely a Definite Crust in later life is pathologic, particularly when there is a definite increase in the incidence of Definite Crust in the period from 16-25 years (Graph 2-A), and an increase in the visible roughness during the same period (Graph 1).

It would appear from its early appearance that this crustiness is in some way related to the presence of the gingivae, for there is a definite decrease in the incidence of a Definite Crust after 25 years (Graph



Graph 3. A. The Frequency of Complete Enamel Loss at the cemento-enamel junction expressed in the percentage of surfaces involved. (4 to each tooth)
B. The Average Amount of Complete Enamel Loss at the cemento-enamel junction in millimeters.



Graph 4. The Amount of Enamel Loss at the cemento-enamel junction when divided into the separate tooth surfaces and also into different groups of teeth. 1 cm.=1 mm.

2-A) and with an increasing depth of the pathologic crevice (Graph 2-B). Note, however, that the total percentage of affected teeth remains the same, the incidence of Slight Crust increasing in direct ratio to the decrease in incidence of the Definite Crust. i.e., By 25 the increase in new crust formation seems to cease for life, for most teeth (90%) are already involved. At this age the attachment of the periodontal membrane is apical to the cemento-enamel junction in 93% of cases[17] and the bottom of the crevice is very near the cemento-enamel junction. Thus, exposure to friction, as discussed above, probably accounts for the change from a Definite Crust to a Shallow Crust.

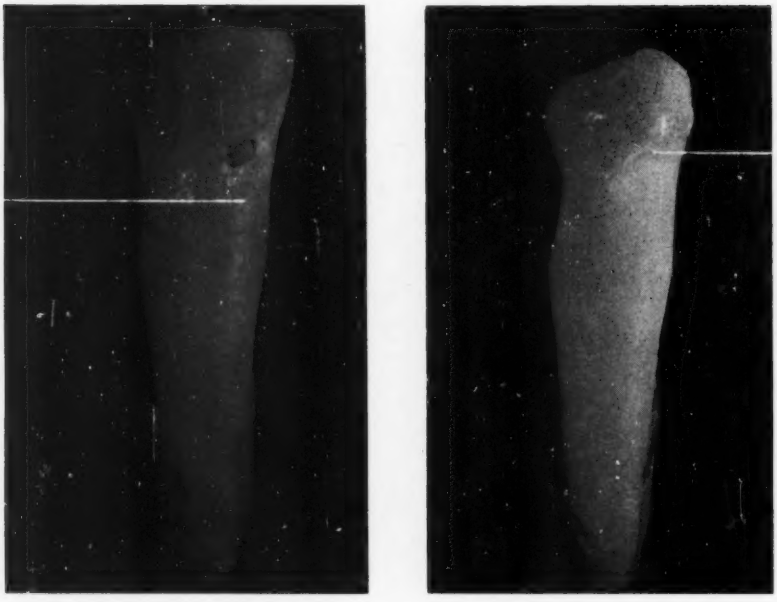
One explanation might be that gingivitis, all too common in early life, so interferes with the normal metabolism of the area that maturation is slowed or stopped; another that the disease process accompanying the gingivitis could actually cause the deterioration. Evidence of some such process will be published later.

In Graph 2-B the teeth were rearranged according to the depth of the pathologic crevice (see legend). Here we find the same general pattern as in Graph 2-A, until we reach the teeth with very deep periodontal ligament detachment. Then we find a continuing decrease in the incidence of the Definite Crust, tempting us to formulate a rule: The deeper the crevice, the slighter the surface crust on the enamel.

In early life, particularly, there is a tendency for these crusty surfaces to occur more frequently on the buccal (15%), slightly less often on the lingual (12%), and still less on the proximal (7% each), which sequence of events recurs constantly in the various stages of our studies.

ENAMEL LOSS AT THE CEMENTO-ENAMEL JUNCTION

Not only is the surface of the enamel lost during the planing process, but in a great majority of these teeth a measurable amount of enamel at the cemento-enamel junction is lost in its entirety. This was determined by recording the distance from the cemento-enamel junction to the apex before



Cemento-Enamel Junction
Before Curettage

Cemento-Enamel Junction
After Curettage

Fig. 1. Enamel Loss at the Cemento-Enamel Junction

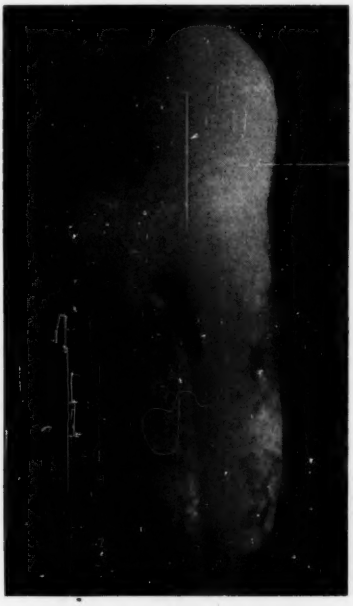
curettage, and again after curettage, the difference representing the amount of loss.

Figure 1 illustrates 2 unusually bad losses of enamel in this area during curettage. It is quite common for this loss to be most marked in one area as shown, but not in the majority of cases. On most, the loss is almost equal on all surfaces, and so small as not to lend itself well to photographic illustration.

Figure 2 represents the characteristic pattern formed, but many variations are possible. The most common pattern is the one illustrated, in which the loss is about equal completely around the circumference of the tooth; the next in frequency, a definitely greater loss on one surface than on the other surfaces, the mesio-buccal being the most common site of this variation; next, 2 surfaces presenting a greater loss than the rest, the buccal usually being slightly greater than the lingual in the young and the lingual greater in the old. Characteristically, but not universally, the illustrated pattern is found in the young—the variations as we study the older teeth.

The Frequency of Enamel Loss is depicted in Graph 3-A. Even at 15 years and younger, while the epithelial attachment is still on the enamel, there is some loss of enamel in 80% of these teeth, this percentage increasing to 94% by 26, and continuing to increase throughout life. Were this normal immaturity of the enamel, there would not be this 14% increase during this period; rather, there would be a decrease as some of these areas completed maturation. Actually, it appears that the enamel matured in only 18% (Graph 2) to 20% (Graph 3) of these teeth, and that if failure to mature is pathology, over 90%

Cemento-Enamel Junction
Before Curettage



Cemento-Enamel Junction
After Curettage

Fig. 1 (Continued)

of enamel surfaces are pathologic at the cemento-enamel junction by the age of 25.

The Amount of Enamel Loss is shown in Graph 3-B, in which the teeth are not only divided into Age Groups, but also are separated into buccal, lingual, and the average of the mesial and distal surfaces. Again we note that the greatest loss is usually on the buccal surface, the second greatest on the lingual, and the least on the mesial/distal surfaces, corresponding to our findings in studying the depth of the pathologic crevice[17] and to our findings on the areas of greatest crustiness, above.

The greatest average loss is only 1.4 mm's and even this slight figure is far greater than that found on most teeth, for it includes many with a major loss as illustrated in Figure 1. The increase of .5 mm from 16 to 25 is probably significant and indicates pathology, for again were it only immature enamel, the loss would be greater in the younger group. The slight variations of .1 to .2 mm's after 25 are probably not significant.

An attempt was also made to determine whether this enamel loss was more common on some groups of teeth than on others. *Graph 4* tells this story. There is a slightly greater loss of enamel on the buccal of the cuspids and bicuspids, but .2 mm is too small a factor for definite conclusions. Clinical memory would seem to tell us that hypersensitiveness, cervical decay, and cervical abrasions are more common on these teeth, but no facts are available. There is a .3 to .4 mm smaller loss on the proximal surfaces of the incisors, an observation which may or may not be significant.

Another factor should be mentioned, but cannot be explained until other findings are published. Not all of this loss of enamel

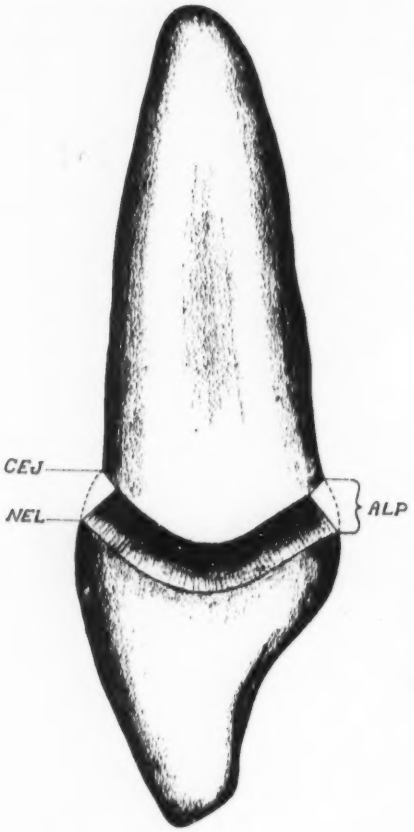


Figure 2. The Characteristic Pattern formed on most teeth when the cervical enamel is curetted with a **sharp** instrument during curettage.

NEL—New Enamel Line after the loss of enamel at the cemento-enamel junction. ALP—Area Lost in Planing. CEJ—Original Cemento-Enamel Junction.

at the cemento-enamel junction is the result of surface softening, deterioration, immaturity, or whatever it may be. There is a process of undermining—a deterioration of the dentin—extending up under the enamel at the cemento-enamel junction in varying degrees, so that the end result on many teeth is not a smooth continuous surface, but a sharp ledge of enamel jutting out from the uninvolved dentin beneath. The experienced periodontist will recognize this characteristic from many unsuccessful attempts to plane the cemento-enamel junction to smoothness in the mouth, a "groove" or "ledge" forming in spite of every precaution.

Still other observations seem to show a relationship between these surface softenings and both smooth surface caries and enamel surface abrasions and erosions, but a description of these will be more understandable when other findings are reported.

DISCUSSION

In a review of 285 text books and articles on periodontia by 198 authors, 91.5% recommend a form of treatment that includes curettage. Most use subgingival curettage alone; many combine it with surgical removal of the overlying tissues; others with packs and drug therapy. When described at all, they recommend the removal of all calculus and organic debris, and the planing of the tooth surface to *smoothness*, emphasis being placed on the use of a *sharp* instrument.

At the chair, in our hands, it has proven impossible to curet the surface of the enamel to *smoothness* with a *sharp* instrument without removing the enamel surface as described in this study on extracted teeth. In fact, we started these studies in an attempt to improve our finger skill in curettage, and blundered into the above findings. Most authors mentioned describe the extreme difficulty of removing calculus, and most instruments designed and widely accepted for subgingival curettage are capable of quite heavy duty. As previously mentioned, it has not been our experience that calculus is common in the early stages of gingivitis or periodontitis, nor is calculus difficult to remove when it is present. We do encounter extreme difficulty, however, in removing the hard crusty enamel surface described earlier.

In other words, it is our opinion that those authors who describe extremely tenacious calculus and the difficulty of remov-

ing it, are in reality removing these crusty surfaces under the false impression that they are removing calculus. For the last 10 years we have recognized this surface crustiness for what it really is and have deliberately removed it as our first step in the treatment of periodontal diseases, be it a mild gingivitis or a late periodontitis. This leads to an orderly, planned procedure, and definitely better results than our previous blind search for spicules or thin layers of calculus which were not there. A discussion of our entire approach to treatment would be out of place without first discussing our findings on the curettage of the root surface, and will be left for later writings. We do wish, however, to emphasize that in our experience, a failure to remove this softened enamel, whether it be a shallow surface or in its entirety, will result in a failure to restore periodontal health, whereas the complete removal of these areas, plus the removal of the root surface deteriorations to be described later, always results in definite improvement and frequently a "cure" of long duration.

SUMMARY AND CONCLUSIONS

Curettement of the cervical enamel of 500 extracted teeth, with a *sharp* instrument, revealed:

1. 21% of the cervical enamel visibly roughened or carious by the age of 25, and 46% after 50 years.

2. A crustiness to the passage of the instrument in 82% of surfaces before 16 years and an increase in this crustiness to over 90% by 25 and throughout life.

3. The crustiness to be more definite in early life.

4. No detectable crustiness on some; a very shallow surface loss on others; a macroscopically visible loss on still others; and on most, a measurable loss of enamel in its entirety at the cemento-enamel junction.

5. The greatest amount of crustiness, and the greatest amount of enamel loss to be on the buccal/labial surfaces; the second greatest loss on the lingual; and the least on the proximal.

Clinically it has proven impossible in our hands to curet the surface of the enamel to smoothness with a sharp instrument in most cases, without removing the enamel surface or even in its entirety at the cemento-enamel junction, and 10 years of deliberately using this procedure as the first step in all periodontal treatment has proven eminently satisfactory.

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The Maintenance Phase of Periodontal Therapy

By RICHARD CHACE, D.D.S., *Orlando, Florida*

THE importance of the toothbrush and gingival stimulation in the maintenance of gingival health following periodontal treatment has been emphasized often in dental literature. There is almost perfect unanimity of opinion as to the value of adequate home care. There have been, however, very few references made to the all important part the dentist must play in preventing the recurrence of periodontal disease. In contrast to the large number of techniques described for the treatment of the periodontal lesion, the maintenance phase of therapy is usually dismissed with a few vague sentences. The neglect of this subject is remarkable since periodontitis has a tendency to recur and in the great majority of cases the excellence of the home care can only retard, not prevent, the reappearance of periodontal breakdown. Since the dentist who treats periodontal disease has a moral and a professional responsibility to maintain gingival health in the treated patient, it might be well to consider the measures he must take to accomplish this end.

PATIENT EDUCATION

The first step in the maintenance phase of treatment is taken when the patient is undergoing active treatment. It is the educating of the patient to accept the fact that he must have expert professional care. after the case has been completed. This first step is very important. The average patient can work up enough interest in his periodontal condition to go through the procedures necessary for restoring oral health, but he will often find excuses for not returning to maintain health. This trait of human nature makes periodontia one of the most exasperating branches of dentistry to practice.

Another point must be put over during the active treatment. The patient must be made to understand that there is a difference between the usual cleaning of the teeth and the preventive treatment. This can be done indirectly by telling the patient that the preventive treatment will take the place of the usual periodic cleaning. It is explained that the cleaning of the teeth is a part of the preventive treatment, but that cleaning alone is inadequate in preventing a recurrence of the original condition.

At the final appointment of active treatment, the patient is given a definite appointment for his first preventive treatment. Within a week a form letter, individually typed, is sent to the patient. It reads as follows:

"Mrs. John Smith
415 Broadway
Orlando, Fla.

Dear Mrs. Smith:

Since we have now finished the treatment of your periodontal condition, I thought I would like to send you a folder that will bring the importance of home care again to your attention. Please read it carefully since your dental future depends to a large degree on how well this advice is followed.

We have set up your first preventive treatment for September 22nd. If you should have any discomfort or bleeding before this time, call us, and we will be happy to see you sooner.

Sincerely yours,"

The enclosed pamphlet is as follows:

"THE HEALED PERIODONTAL PATIENT

"The patient who has had successful treatment for pyorrhea is in very much the same category as is the patient who has been discharged from a tuberculosis sanatorium. In both cases the patient is cured of the disease. In both cases the

disease is likely to recur unless the patient takes good care of himself. It is important, therefore, that the healed periodontal patient realize what steps he must take in order to maintain his hard-won oral health.

"The first step is the careful following of the home care routine that was taught during the treatment of the pyorrhea. There is a tendency for the patient to spend less time and effort on his tooth brush technique after treatment has been completed. Every periodontist has seen patients whose teeth were lost within a few years because they either could not or would not follow an adequate home care program. On the other hand an apparently hopeless case can often preserve his teeth for years by being particularly skillful and faithful with his toothbrush. It can be said that the person with a tendency for pyorrhea requires at least twice as good home care as does the average person.

"The second step is the cooperation of the patient with the periodontist by coming in regularly for his preventive treatments. These preventive treatments are the treatments that the dentist must give the healed patient to prevent periodontal disease from returning. A treatment consists of a prophylaxis, or cleaning, the checking of home care, the careful exploration and curettage of the gum crevices, and the checking of the bite. Any minor trouble that might be starting is taken care of at this time. These preventive treatments take the place of the usual cleaning which is inadequate in most cases to maintain oral health. The time between each treatment and the extent of a treatment varies with the individual patient.

"Success in periodontia is always relative. The object of dentistry is to eliminate infection, restore function, and to preserve the natural teeth for as long as possible. The ideal would be for no person to ever have to wear artificial dentures. While this ideal cannot always be attained, it may often be approached by a partnership of the dentist and patient. With the completion of active treatment, the role of the patient becomes more and more important. To a large degree it is up to him."

The appointment routine for the preventive treatment varies considerably. There are many different types of patients in any periodontal practice. Some have a marked tendency to collect supra-gingival calculus and stain. Others have mouths that are almost self-cleaning. In the case of the patient who had an advanced periodontitis complex or periodontosis, a preventive treatment is in reality a continuation of active treatment. The time between appointments is now measured by months rather than by days. There are also healed cases of periodontitis that need only careful prophylaxis to maintain oral health. It is desirable, therefore, to tailor the appointment to fit the patient, both in regards to the frequency and the type of appointment.

THE MISUSE OF THE DENTAL HYGIENIST

A word must be said in regards to the misuse of the dental hygienist. In many instances the entire preventive treatment is given by the hygienist, the dentist appearing only for a few minutes at the end of the appointment to greet the patient and to casually examine the mouth. While the financial advantages of relegating the preventive treatment to the hygienist are obvious, the practice must be condemned as dishonest. In the first place instrumentation, even if the hygienist possessed enough skill to do it adequately, is only a part of the preventive treatment. A thorough examination of the occlusion and of the gingival crevice is necessary, and these operations require both time and care. It must be remembered that a pathological deepening of the gingival crevice may be most inconspicuous. This is particularly true when the patient is following his home care regime carefully. It must be remembered also that in many cases the etiological

factors are unknown and that they are presumably still operating, even though the periodontal lesion may have been eliminated. A periodontal breakdown around a previously unaffected tooth is often found during the maintenance phase. The preventive treatment should be designed not only for the prevention of the recurrence of old pockets but for the very early recognition and treatment of new ones. With this concept, the role of the dental hygienist, useful as she may be, must be a minor one to that of the dentist on whose shoulders the responsibility lies.

THE PREVENTIVE TREATMENT

The average recall appointment begins with the casual examination of the mouth by the dentist. The operator merely wishes to get a general idea about the excellence of the home care and of the general condition of the mouth. The patient is then turned over to the hygienist for a thorough prophylaxis. If instruction is needed with the toothbrush in some area the hygienist gives it at this time. The dentist then begins his part of the treatment. He goes over the entire mouth with a very sharp set of curets, using an extremely light touch. It is most important that every gingival crevice be probed, either with the curet or with a pocket probe, to be sure that the depth is not increasing. During this procedure constant reference is made to the original chart and to the notes on the résumé of the case after active treatment was completed. (Fig. 1.) Any deviation from the normal is charted on the small chart pasted to the rear of the record. (Fig. 2.) It must be remembered that it is very easy in the original treatment to overlook porous areas in the cementum and small deposits of calculus. The preventive treatment gives the operator a chance to perfect his result. The ivory-like smoothness of properly prepared cementum does not tend to collect sub-gingival calculus but very fine deposits are often encountered and should be removed. The properly sharpened curet can also detect abnormalities of the soft tissue wall. Should granulation tissue be encountered, it must be removed. This latter operation is almost painless and is done with the reverse edge of the curet.

It has been asked why the hygienist is used at all if the dentist must go over the mouth this carefully. It will be found that it is much easier to operate in a mouth free of supra-gingival calculus and stain. The operator can usually complete the light curettage in about ten minutes, although some mouths will take more time.

The occlusion is then checked. Almost invariably some premature point of contact will be found, either in centric or in one of the lateral movements. This is due to the fact that uneven wear is the rule rather than the exception, and that the teeth often shift their position slightly. On succeeding preventive treatments it will be found that less and less work with the occlusion will be necessary.

If nutritional supplements have been prescribed, the patient is questioned about his faithfulness in taking the prescriptions. It will be found that the average patient does not follow nutritional advice for a long enough period. He must receive encouragement and continued advice if this phase of treatment is to prove any real help. Here again the résumé of the case on the back of the treatment record is of great value. A busy practitioner will not usually take time to read all of the records over before the patient comes in. The résumé is in reality the abstract of the active treatment and will prevent the dentist from making many embarrassing mistakes.

TREATMENT OF MINOR DEFECTS

It is very rare that a patient will present himself for a preventive treatment with a mouth in perfect condition. In successful treatment such deviations from health are usually minor. It is the purpose of the preventive treatment to discover these minor defects and to treat them so that they will not become major problems.

It is occasionally necessary to call the

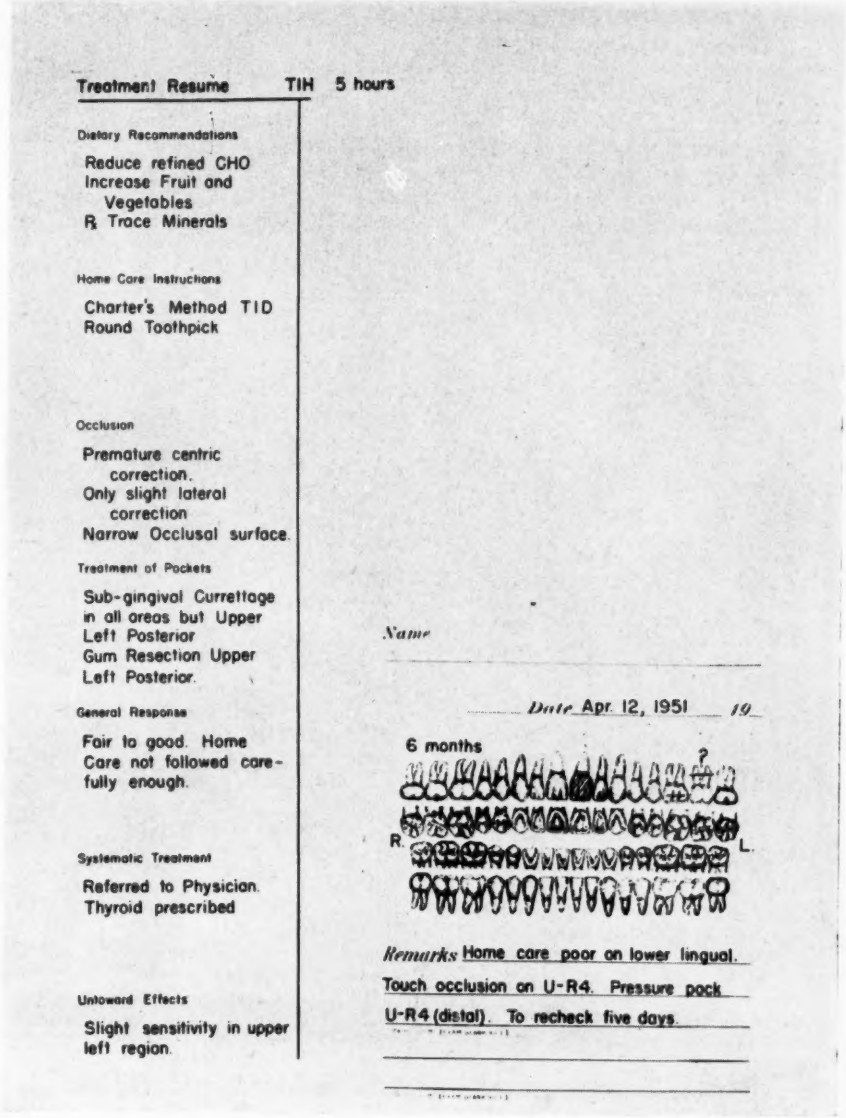

Fig. 1. Résumé of active treatment.

patient to task if his home care efforts have been inadequate. While the approach to this subject may vary with the patient, it should be always made firmly. The patient should be shown his shortcomings with disclosing solution and be made to understand in no uncertain terms what the results of his poor home care are. Another ap-

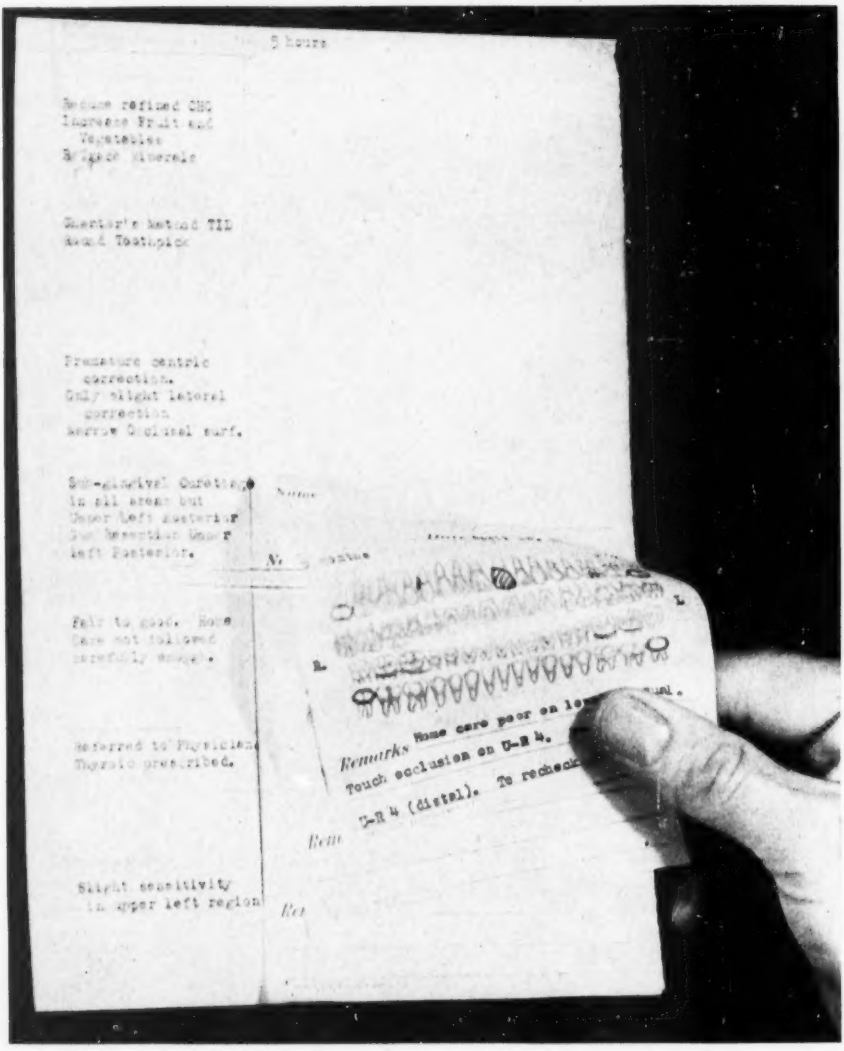

Fig. 2. The résumé of active treatment is given in the left column. A small examination slip is pasted on the card for each preventive treatment. Only the abnormalities are recorded. The record of the preceding preventive treatments can be easily referred to. In practice the notes on the treatment are written in pencil while the patient is in the chair.

pointment for the checking of the home care efficiency should be made even though the treatment can be completed in the reserved appointment.

The patient whose toothbrushing habits are good should not be taken for granted. He must be complimented and inspired to continue his efforts. The effective care of the mouth is a drudgery for many patients and they need all the encouragement you and your hygienist can give them.

Slight sensitivity of the necks of the teeth to instrumentation is rather commonly encountered in preventive treatments. It is often found when the patient notices no discomfort in either eating or brushing. The dipping of the instrument head in 8% zinc chloride before applying it to the tooth will usually make the operative procedure more comfortable.

Exquisite areas of sensitivity are by no means uncommon. The patient is bothered in his home care efforts and is often obliged to keep cold food and liquids away from such areas. The vigorous use of a port-polisher dipped in either formaldehyde or 50% zinc chloride will usually relieve such points, although it is often necessary to repeat such applications. The use of a surgical pack for a few days following the port-polishing will also increase its effectiveness. Merritt[1] advocates the covering of such areas with a thin mix of oxyphosphate cement, and allowing the cement to remain in place for several weeks. Occasionally this will be found to be necessary.

A sensitivity under the margin of the gum tissue presents another problem. The use of a paraformaldehyde pack as advocated by Orban[2] will not only reduce the surplus tissue but will most effectively treat the sensitivity.

In all cases of sensitivity the use of paraformaldehyde tooth-powder will be helpful. It should also be burnished into the sensitive areas with a round toothpick. An effective formula is as follows:

Dicalcium phosphate, drachms 15
Sodium oleate, drachms 12
Oil of lemon, min. 12
Oil of pimento, min. 7
Oil of peppermint, min. 12
Saccharin, grains 1½
Paraformaldehyde, 2 per cent by weight

Occasionally a patient will complain that from time to time all of his teeth become very sensitive and that this condition may persist for several days. We have found that the general sensitivity that occurs suddenly is usually due to a lowering of the patient's general resistance. A common cold or a gastro-intestinal upset will often be preceded by this type of sensitivity. The reduction of refined carbohydrates, the deletion of cold solids and liquids from the diet, and the use of the paraformaldehyde toothpowder will usually hasten the return to normal.

Areas of inflammation are often encountered even in the mouth that has responded well to treatment. Such areas are usually due to some lapse in the toothbrushing technique for that part of the mouth. It is quite important that the error in the brushing technique is corrected. The inflamed area is treated with super-oxol.

Occasionally, examination will disclose the recurrence of a previously eradicated pocket. In this case it is important to know whether the increased crevice depth is due to the hypertrophy of the gingiva or to breakdown of the deeper periodontal tissue. The former is usually due to calculus formation or the accumulation of soft deposits on the teeth with resulting gum irritation. Instrumentation, application of super-oxol, and correction of the deficiency in home cleansing will often take care of this type of recurrent pocket. A breakdown in the deeper tissue may be the result of an overlooked occlusal trauma.

Individual recurrent pockets of some depth may be treated with potassium hydroxide paste as advocated by Gratzinger.[3] It is important not to try to destroy more than two or three millimeters of tissue at a time with this paste. It is a relatively painless method of tissue destruction, and it obviates the necessity of anesthesia and surgery.*

*KOH	9.0 gm.
Magnesium Carbonate	.3 gm.
Thymol	.3 gm.
Oil of Peppermint	.3 gm.
Glycerine Q. S. Stiff Paste	

Should the recurrence of pockets be more generalized, the use of pressure packs as advocated by Robb[4] may be considered.

The pack should be mixed quite stiff and considerable pressure must be used in placing it. The pack is most effective when the tissue to be reduced is in the interproximal area. Occasionally a second application of pack will be found to be necessary.

If the use of the caustic paste is found necessary, the patient should be asked to return in four or five days so that the treated area may be observed. The pressure packs are usually removed in two or three days and the mouth re-examined in a week. It is also well to check the patient again if much inflammation was present at the time of the treatment. Such short check-ups tend for much better patient control and make the maintenance of the case more sure.

It has been observed that there is often a gradual regrowth of gingival tissue following a gum resection. This regrowth occurs without inflammation and it usually takes at least a year or two to occur. The gum tissue is well keratinized and appears quite normal. The gingival crevice, however, increases in depth since little reattachment takes place. The writer regards this regrowth as a healthy occurrence provided the gingival crevice does not exceed 3-4 mm. Such cases require special care in the preventive treatment, however.

There are other types of cases where the gingival depth is greater than is generally regarded as normal. Sometimes pockets cannot be completely eliminated during treatment. Every periodontist has a few such compromise cases that he carries along in his practice. For all patients with deep gingival sulci a special type of preventive treatment is scheduled. The majority of the appointment time is reserved for the dentist since more time is required to curet the deepened crevice and the hygienist cannot be expected to perform this difficult operation.

Quite often the active treatment of an intra-bony pocket or pockets may continue for months after the rest of the mouth is in the purely maintenance phase of therapy. In spite of the newer techniques, the closure of intra-bony pockets is uncertain and it often occurs a little at a time. Such pockets are thoroughly recuretted and sealed at the end of the preventive treatment, and the patient is cautioned not to brush the area for several days.

The stain forming patient is often a source of needless discouragement to the periodontist. Except for the unesthetic aspect of stain it seems to do little harm, and some patients find it impossible to prevent its formation. The patient must be told of the unimportance of stain formation so that he will not be discouraged in his home care efforts. The heroic removal of all stain by the dentist on each recall visit is to be frowned on. Stain can be completely removed from root surfaces only when cementum is removed also.

RADIOGRAPHS

There are no "hard and fast" rules about taking radiographs. The patient with advanced disease or with the complex type of periodontitis should be x-rayed often. A full mouth set should be taken about every two years in the average case. It is best for two appointments to be given for this. On the first the exposures are made. The preventive treatment is given on the second and the mouth is completely re-charted. In between appointments the new and the old series are carefully compared.

TIME BETWEEN PREVENTIVE TREATMENTS

The time between recalls varies with the patient and with the length of time since the active treatment was completed. The writer has found that the first year after the active treatment is the most crucial time in maintaining health. The mouth seems most likely to break down during this period. For this reason the patient is seen at least three times during the first year. The first appointment for preventive treatment is usually given three months following the last active treatment, although some cases are seen within two months. From then on it is a matter of the need of

the individual patient. It is felt that the patient should be seen on recall before any regression of the case could take place.

SUMMARY

The maintenance of the healed periodontal patient has been a subject that has been neglected both in the text-books of periodontia and in the current literature. If the saving of teeth and the preserving of health are criteria of importance, however, this phase of therapy should be carefully considered and a definite routine must be worked out for office practice. The hygienist is an indispensable aid to the periodontist in preventive treatments, but the dentist himself must fulfill his professional responsibility to the patient.

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Clinical Research Needed for Erosion-Abrasion*

By CHARLES F. BODECKER

THE cause of the condition known as erosion of the cervical areas of the teeth is still a vexing problem. Not only does this condition result in discomfort to the patient due to temperature changes, but it necessitates the placing of fillings in highly visibile areas. The problem is one which dentistry should obviously solve.

Cervical erosion appears to be a disease of modern life. Generally it is ascribed to incorrect tooth brushing habits. Undoubtedly, deep horizontal notches in easily accessible areas are often due to an incorrect cross-bow tooth brushing technic. However, not all lesions are in the form of notches; some are so irregular in outline that they cannot be caused in any manner by the use of a tooth brush (Fig. 1). Further, there are cases on record of erosion lesions occurring on the lingual and even proximal tooth surfaces. One such case was recently observed by Dr. Arthur H. Merritt. As tooth brush abrasion cannot be the cause of lesions in areas inaccessible to forceful brushing, another factor has long been advanced by a minority of dentists, *viz.* locally exuded mouth acid. The writer is among this minority (Local Acidity: A Cause of Dental Erosion. Progress report of the Erosion-Abrasion Committee of the New York Academy of Dentistry, *Annals of Dentistry*, 4:50-55, September 1945). A third possibility is suggested by Dr. Benjamin Kornfeld that erosion lesions may be caused by abnormal pulp activity.

The investigation conducted by the group referred to in the above progress report established the following results: The fluid under the cervical gingivae was tested in 469 teeth with blue and red litmus paper. Of the 132 teeth having erosion lesions, 60 per cent showed an acid crevicular fluid, 18 per cent were neutral and 22 per cent were alkaline. Of 151 sound teeth, on the other hand, only 18 per cent tested acid, 22 per cent were neutral and 60 per cent were alkaline. This definite trend indicated that acid is more often present around erosion lesions than around sound teeth.

*This article, since it is of interest to all dentists, is being published simultaneously in the October 1951 issue of the New York State Dental Journal.

Differentiation Between

	Abrasion Lesions	*Erosion-Abrasion Lesions*
1. FORM	Notches, walls roughly at right angles to tooth surface; more rarely as horizontal grooves (Fig. 1-A).	Shallow, saucer-shaped (Fig. 1-E).
2. LOCATION	(a) Usually close to gingival margin on labial or buccal surfaces. Grooves may be on middle of tooth.	(a) Always commence at gingival margin unless caused by an acid buccal or labial gland.
	(b) Occur on prominent tooth surfaces.	(b) Prominence a minor factor.
	(c) Areas easily accessible to tooth brush; lesions are on left side in a right-handed person and vice versa.	(c) Distributed more evenly on left and right side of mouth.
	(d) Occur on adjoining teeth if equally prominent.	(d) Adjoining teeth not necessarily affected.
	(e) Somewhat more common on maxillary than mandibular teeth.	(e) Definitely more common on maxillary teeth.
3. SURFACE OF LESION	High glass-like luster common also to all tooth surfaces.	Clean but not as highly polished.
4. SENSITIVITY	Not sensitive to touch; occasionally to temperature changes.	Often highly sensitive to touch and temperature changes.
5. SUSCEPTIBILITY TO CARIES	Has not been observed. Mouth almost caries-free.	On rare occasions may become carious.
6. GINGIVAL CONDITIONS	Firm, pink, no trace of gingivitis.	Usually normal, only occasionally trace of gingivitis.
7. TYPE OF PATIENT	(a) Much more common in males than females.	(a) Somewhat more common in males.
	(b) Determined, vigorous, generally "live wire" type; not phlegmatic; tendency to stocky, powerful build. Often have mania to keep teeth clean and tendency to occlusal neurosis.	(b) Nervous, high strung, mercurial type, thin rather than stocky build; also far from phlegmatic; place less emphasis on oral hygiene.

However, further observations should be made regarding the conditions under which these lesions develop. Apparently, there are two distinct types of these lesions, sometimes termed cervical wasting (W. D. Miller, Hirschfeld), each having a different etiology. One appears to be caused purely by abrasion (tooth brush, loose denture clasps, incorrect use of tooth pick, etc.) and the other by an acid crevicular fluid plus abrasion. As to the latter type, it is believed that the lesion forms when the acid causes a slight superficial softening of the enamel and dentin which is then brushed away by the tooth brush so that the surface of the lesion always remains highly polished and hard.

The following table represents the conclusions of the writer as a result of clinical observations over a long period of years:

The problem of erosion-abrasion cannot be solved in the scientific laboratory. Its cause and prevention can only be determined by the dental practitioner, and the periodontist is in a position to make the most definite contribution as he is most interested in gingival tissue and tooth cervices.

A suggested approach to the problem, which may be of assistance to the investigator, is as follows:

1. Select patients with erosion-abrasion lesions.

2. Chart devital teeth, if any, and take usual case history including caries index, tooth brushing habits, right or left handed,

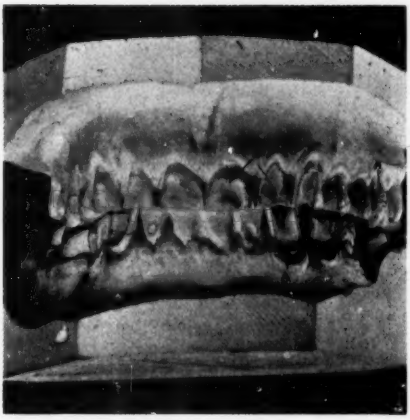

Fig. 1. Patient 46 years of age showing exceptional and extensive erosion (E) on both sides of the mouth, predominating slightly on left. The lesion on left maxillary central and lateral incisors, for instance, could not have been formed by tooth brush abrasion. The left lower cuspid and upper first molar, however, show deep horizontal grooves (A) probably caused by tooth brush abrasion. Teeth have been worn down markedly by occlusal neurosis. Lesions outlined in dots.

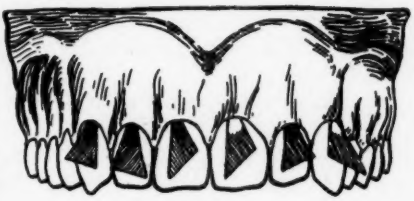

Fig. 2. Shows manner of testing pH of crevicular fluid with pHydrion paper; presence of acid indicated over maxillary central incisor and cuspid tooth.

kind of dentifrice (tooth powders), temperament of patient (nervous, phlegmatic, determined, forceful), habits (sucking lemons), tendency to occlusal neurosis, etc.

3. Chart location of lesion and determine which are sensitive to dental explorer, temperature or chemical changes.

4. Cut pHydrion paper into small triangles. (This gives more exact pH determination than litmus paper; obtainable through Micro Essential Laboratory, Brooklyn, N. Y.)

5. Insert one point of paper under the gingiva near lesion to determine pH reaction (Fig. 2) and enter on chart.

6. Test the reaction of the crevicular fluid of neighboring teeth having no erosion lesions.

7. Note condition of gingivae where lesion is present and absent.

8. Take pH of saliva at parotid duct and under the tongue, noting its physical character (thin, watery, or thick, ropey).

A chart for recording these observations will be made available by the Editor to any investigator requesting one.

Perhaps a thorough survey along these lines will lead to the discovery of the cure of this disease and the means for preventing it.

(Continued from page 224)

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Editorials

The Unsolved Problem of Dental Erosion-Abrasion

Among the more or less common lesions affecting the teeth is one commonly referred to as toothbrush abrasion or, more accurately as erosion-abrasion. The exact nature of these lesions and their cause is at present far from clear.

That the toothbrush and the way it is used, is a factor there can be little doubt. Another factor too often overlooked, is the abrasive effect of the dentifrice. Since these vary in their abrasiveness it is not at all improbable that the dentifrice plays an important part in many of the cases seen in daily practice.

Several years ago the writer was asked by a colleague if he had noticed that patients using a certain well known dentifrice showed marked buccolabial abrasion of their teeth. A subsequent study of cases seen in practice confirmed this observation.

Another factor and one less well understood is the possibility in some cases of an acid exudate from the gingival sulcus causing a superficial softening of the cervical enamel, thus making it more easily removed by the combined action of the toothbrush and dentifrice.

Occasionally erosion may also occur on the buccolabial surfaces of the teeth midway between the cervical margin and occlusal surface, possibly caused by an acid secretion from labial or buccal glands in contact with the affected teeth. Fruit acids, candy, etc., may, under certain circumstances produce lesions similar to erosion. Hirschfeld* refers to cases of this type which he calls "chemical erosion" in which the "wasting . . . was generalized except for a narrow strip of enamel, close to and parallel with the gingival margin" which in some cases he refers to as being caused by fruit acids such as oranges and lemons. The fact that the enamel parallel with the gingival margin was *not* affected, would seem to indicate that the solvent, whatever it may have been in these cases, did not have its origin in the gingival sulcus.

Very rarely one sees a type of erosion-abrasion characterized by a gradual loss of tooth substance in areas not easily accessible to the toothbrush. A case recently seen by the writer showed considerable flattening of the lingual surfaces of several teeth, plus deep polished grooves on proximal surfaces.

That there is great need for more research in this field there can be no doubt. And who could be more concerned in finding a solution to the problem than the periodontist who sees these cases in his office daily? In this research he can be most helpful since it is a clinical rather than a laboratory problem.

The reader is referred to an excellent article by Dr. Bodecker on the subject of erosion-abrasion in this issue of the Journal and is urged to cooperate with

*The Toothbrush: Its Use and Abuse. Dental Items of Interest Publishing Company 1939, 310-320.

him in an effort to find the answer to this perplexing question. A chart for recording one's observations can be had from Dr. Bodecker on request.

A. H. M.

Michigan Workshop A Success

The School of Dentistry and the School of Public Health of The University of Michigan offered an inservice training course for the "Appraisal of Present Scientific Information Concerning Periodontal Disease" at Ann Arbor, September 10 through 15, 1951. This course proved to be very popular (it was oversubscribed), and much good should come from it.

The Workshop was carefully planned by Dr. Kenneth A. Easlick, who served as Chairman and Director of the course, assisted by Dr. Harry E. Miller, Director of Continued Education at the University of Michigan, and Dr. Henry F. Vaughan, Dean of the School of Public Health. Members of his advisory committee included a representative of the American Dental Association, Dr. Allen O. Gruebbel, and the following representatives of state departments of health: Dr. David B. Ast, New York; Dr. John E. Chrietzberg, Illinois; Dr. Paul Cook, Louisiana; Dr. Hugo Kulstad, California; Dr. William H. Rumbel, Virginia; Dr. Carl L. Sebelius, Tennessee; Dr. Roy D. Smiley, Indiana; and Dr. Fred Wertheimer, Michigan. Also included were members of the faculty of the University of Michigan School of Dentistry, Dr. Donald A. Kerr, Dr. Hilda M. Rankin, and Dr. Clarence J. Velz; and Dr. H. B. G. Robinson of Columbus, Ohio, and Dr. Leo J. Schoeny, New Orleans.

As is usually done in workshops, lectures were held in the mornings and evaluation committees met after lunch. "Students" attending the course were assigned to one of the following Evaluation Committees: (I) Behavior of the Gingival and Supporting Tissues, Chairman: Joseph L. Bernier; (II) Local Factors in Periodontal Disease, Chairman: Thomas J. Hill; (III) Systemic Factors in Periodontal Disease, Chairman: H. B. G. Robinson; (IV) Treatment of Periodontal Disease, Chairman: Henry M. Goldman; (V) Prevention of Periodontal Disease, Chairman: Paul E. Boyle. These committees prepared and presented reports for consideration on the final day.

Discussions were held on the behavior of oral tissue, etiologic factors in periodontal disease, hyperplastic gingivitis, advanced periodontal disease, and management of periodontal disease. Most of the subjects were carefully prepared and well presented, and everyone agreed that the week was an informative one.

In retrospect, the following general comments seem in order:

1. The conference emphasized the need for more clinical and laboratory research not only to learn more about the etiology, pathology, treatment and

prognosis of periodontal diseases, but to establish scientifically much of what is already believed to be true.

2. There is need for more study in the field of physiology of the periodontium.

3. There is need for more information regarding prevention of periodontal diseases. Very little emphasis was placed upon prevention of calculus depositions at this conference, for example, simply because very little is known.

4. Methods must be developed to allow evaluation of the state of health and disease of the periodontium in large groups of individuals.

Undoubtedly the papers and discussions given at the conference will be published, as they included much of what is and is not known in the field of periodontology. The American Academy of Periodontology and the dental profession owe the University of Michigan a debt of gratitude for organizing and presenting this fine course which attempted to appraise in a critical fashion the scientific information which is available concerning periodontal disease.

Announcements

UNIVERSITY OF TORONTO

The Faculty of Dentistry, University of Toronto announces a series of short intensive courses for dentists during the 1951-52 session. These courses will be arranged and conducted by the staff of the Faculty of Dentistry with the cooperation and financial support of the W. K. Kellogg Foundation of Battle Creek, Michigan. When deemed advisable, outstanding visitors will be included among the lecturers and clinicians.

Periodontics—October 29-November 2, 1951. This course will include consideration of the nature of periodontal disease, examination and case management, pocket therapy, oxygen therapy, metabolic influences of the periodontium, significance and correction of occlusal function, gingival recession, pocket surgery, applications of principles of periodontal practice to operative and prosthetic procedures.

The course will be under the general directions of Doctors H. K. Box and C. H. M. Williams.

Tuition Fee $75.00 Enrollment Limit 14

Dental Public Health Conference—November 20, 21, 1951
9:00 a.m.-5:00 p.m. Enrollment limit 15.

Dental Radiology—February 25-March 1, 1952
9:00 a.m.-5:00 p.m. Enrollment limit 12.

Endodontics—March 10-14, 1952
9:00 a.m.-5:00 p.m. Enrollment limit 8.

Periodontics—April 28-May 2, 1952
9:00 a.m.-5:00 p.m. Enrollment limit 14.

Other courses will be arranged as the demand indicates. Those interested are asked to inquire respecting enrollment. Please address inquiries to: The Dean, Faculty of Dentistry, University of Toronto, 230 College Street, Toronto 2-B, Ontario.

1951-52 STUDENT ESSAY CONTEST OF THE AMERICAN ACADEMY OF PERIODONTOLOGY

To promote interest in periodontia among graduate students, the American Academy of Periodontology is sponsoring an annual essay contest. This contest is in two general parts:

1. *Local Contest in each school.* Each year the American Academy of Periodontology wishes to award a year's subscription to the Journal of Periodontology to the undergraduate student who writes the best paper on periodontia in each college. Each faculty of each college can make its own rules and regulations and select the winning paper. The winner's name should be sent to the Editor of the Journal before May 15 of each year.

2. *National Contest.* Each year one paper from each school can be submitted to the Undergraduate and Postgraduate Education Committee of the American Academy of Periodontology on or before April 15. The three best papers will be awarded the following prizes:

First Prize—$50
Second Prize—$30
Third Prize—$20

These papers or abstracts of them may be published in the Journal of Periodontology as the best undergraduate papers submitted in the year.

The American Academy of Oral Pathology will hold its Sixth Annual Meeting at the Stevens Hotel, Chicago, on February 3, 1952.

The program will include discussions on the Physiology of Bone, Bone Behavior, and Periodontal Disease, Dermatological Diseases affecting the Oral Cavity, Studies of Oral Carcinomas, Dental Anomalies, and Gingival Enlargement.

All interested dentists are welcome to attend the meetings.

Hamilton B. G. Robinson,
Chairman, Publicity Committee

EIGHTH ANNUAL DENTAL SEMINAR—PALM SPRINGS, CALIF.

Two hundred dentists, including top dental-medical researchers, will be in attendance at the Eighth Annual Dental Seminar set for the Desert Inn, Palm Springs, California, October 28 through November 1, 1951.

Advance registrations, according to an announcement by Dr. Hermann Becks, seminar president, indicate a capacity turn-out for the five-day study sessions.

A Paradentopathies Workshop will be a major feature of the professional gathering this year, with the entire attendance divided into special study groups devoted to various phases of the problem.

Included in the program, to be discussed and analyzed at the meeting are the following important topics:

Practical physiological lung problems in the sedentary professional man.

Cancer of the oro-pharynx and respiratory system.

The role of infection in periodontal disease.

Etiological factors in periodontal disease.

Studies on the defense mechanisms of the mouth and mucous membranes.

Nutritional disturbances in childhood, with particular emphasis on the celiac syndrome and pancreatic insufficiency.

The clinical laboratory in a children's hospital.

The structure and properties of tooth surfaces as studied by optical and electron microscopy.

Electron microscopy of dental tissues—including enamel, dentin, cuticle, epithelial attachment, etc.

Protein deficiencies via soil deficiencies.

Some new concepts concerning the role of sugar in dental caries.

The putrefaction of proteins in relation to periodontal disease.

Seminar lecturers will participate in a Round Table Forum covering the application of the subjects studied to dental practice, which will conclude the 1951 sessions.

The courses in periodontology listed below will be presented throughout the academic year of 1951 and 1952 at Tufts College Dental School. Information may be obtained by directing correspondence to the Director of the Division of Graduate and Post-graduate Studies, Tufts College Dental School, 136 Harrison Avenue, Boston 11, Massachusetts.

Periodontology

Each Monday for the Twelve Weeks from
Oct. 8, 1951 to Jan. 14, 1952
Feb. 25th to May 19, 1952

Tuition $175.00

DR. IRVING GLICKMAN AND STAFF

Occlusal Equilibration
Each Monday for the Five Weeks from
Jan. 21 to Feb. 18, 1952
Tuition $100.00
DR. IRVING GLICKMAN, DR. EDWARD T. FISCHER AND STAFF

Surgical Pathology of the Oral Cavity
and Adjacent Structures
Each Thursday afternoon for the Eight Weeks from
Oct. 4 to Nov. 29, 1951
Tuition $100.00
DR. IRVING GLICKMAN AND STAFF

Periodontology
Nov. 5 to Nov. 9, 1951
June 2 to June 6, 1952
Tuition $100.00
DR. IRVING GLICKMAN, INSTRUCTOR

Periodontology
May 12 to May 16, 1952
Tuition $100.00
DR. OLIN KIRKLAND, INSTRUCTOR

Advanced Course in Periodontology
Nov. 26 to Nov. 30, 1951
Tuition $150.00
DR. IRVING GLICKMAN AND STAFF

Occlusal Equilibration
April 14 to April 18, 1951
Tuition $100.00
DR. IRVING GLICKMAN, DR. EDWARD T. FISCHER AND STAFF

Program of the Thirty-Fifth Annual Meeting
of the
American Academy of Periodontology
Washington, D. C.

Thursday, October 11, 1951

A. M.

8:30- 9:30 Registration

9:30 Invocation
Secretary's Report
Committee Reports
Presidential Address—Dr. Hunter Allen

11:00-11:30 "A Method for Post-Gingivectomy Pack Stabilization" (A Preliminary Report)—J. S. McKenzie

11:30-11:50 "Failure in Periodontal Therapy" (Case Report)—Frank E. Buebe

11:50-12:00 "An Interesting Case of Bite Habit" (Case Report)—M. Monte Bettman

P. M.

2:00- 3:00 "The Development of Periodontal Pockets"—James Nuckolls

3:00- 3:30 "The Elimination of Abnormal Occlusal Stress as a Factor in Healing and Repair of the Periodontal Structures" (Case Report)—J. C. Westbrook, Jr.

3:30- 4:30 "Development in Experimental Pathology which Pertain to the Periodontal Field"—Irving Glickman

4:30- 5:00 "Advanced Periodontosis in a Four and One Half Year Old Boy" (Case Report)—Edward L. Ball, Jr.

Friday, October 12, 1951

A. M.

9:00- 9:30 Report of Past Presidents' Dinner

9:30-10:30 "Permeability of Root Structures Tested with Radioactive Materials"—William W. Wainwright

10:30-11:00 "Dietary Analysis in Routine Office Practice"—Arvin William Mann

11:00-12:00 "Oral Bacteria and Oral Disease"—David T. Smith

P. M.

2:30- 3:00 "Some Recent Concepts of a Connective Tissue Ground Substance and Their Implications in Dental Disease"—Milton B. Engle

3:00- 3:30 "Report of Case of Acute Fulminating Suppurative Periodontitis" (Case Report)—Willa Yeretsky

3:30- 4:30 "The Role of Saliva in Calculus Formation"—S. Wah Leung

4:30- 5:30 "Occupational Bruxism and Its Relation to Periodontal Disease"—John Ingle

Saturday, October 13, 1951

A. M.

9:00-11:30 Symposium: "The Management of Periodontal Pockets"
Joseph L. Bernier, Moderator
Walter Leabo
Charles Williams
Olin Kirkland
Donald Kerr

11:30 Business Meeting and Election of Officers

TABLE CLINICS

Dr. Rowe Smith 405 East Fifth Street Texarkana, Arkansas-Texas	"Diagnosis and Treatment of Periodontal Disturbances Associated with Buckling of Lower Anterior Teeth."
Dr. Justin D. Towner 1206-11 Exchange Building Memphis 3, Tennessee	"Instruments and Instrumentation."
Dr. Edgar James 1802 David Broderick Tower Detroit 26, Michigan	"Functional Analysis of Occlusion."
Dr. Allison G. James 409 North Camden Drive Beverly Hills, California	"Conservation of Supporting Structures in Multiple Tooth Replacements."
Dr. Bernard S. Chaikin 29 Commonwealth Avenue Boston, Mass.	"Indications for Gingivectomy and Subgingival Curettage."

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